A STUDY ON EVALUATION OF MOTOR, COGNITIVE AND BEHAVIORAL MANIFESTATIONS OF BASAL GANGLIA INFARCTS

Dissertation submitted to

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for the award of the degree of

DM (NEUROLOGY) – BRANCH – I

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

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CERTIFICATE

This is to certify that the Dissertation entitled, **"A STUDY ON EVALUATION OF MOTOR, COGNITIVE AND BEHAVIORAL MANIFESTATIONS OF BASAL GANGLIA INFARCTS"** is the bonafide record work done by Dr.G.Gnanashanmugam, under our guidance and supervision in the Institute of Neurology, Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai, submitted as partial fulfilment for the requirements of D.M. Degree examination Branch I NEUROLOGY, AUGUST 2011, under The Dr.M.G.R. Medical University, Chennai.

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DECLARATION

I solemnly declare that this dissertation titled "**A STUDY ON EVALUATION OF MOTOR, COGNITIVE AND BEHAVIORAL MANIFESTATIONS OF BASAL GANGLIA INFARCTS"** is done by me in the Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of Prof. Dr. R.M. Bhoopathy, M.D., D.M., Professor of Neurology, Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to the Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfilment of the university requirements for the award of the degree of D.M.Neurology.

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CONTENTS

INTRODUCTION

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Isolated basal ganglia infarcts are rare. Basal ganglia infarcts are often associated with infarcts in other structures like cerebral cortex, thalamus and fronto parietal white matter. Clinical consequences of basal ganglia infarcts are often masked by infarcts in other areas. When basal ganglia infarct extends into the adjacent internal capsule, more concern and priority will be given to the hemiplegia and associated behavioral and cognitive features may be overlooked.

Several studies have been conducted to analyze the clinical consequences of isolated basal ganglionic infarcts. Most of the studies were conducted in several countries outside India. So, we decided to analyze the clinical consequences of isolated basal ganglia infarcts in our patient population.

Normal functions of many neurological structures were identified by the consequences of destruction of these structures. The pathologies that damage the human brain are rarely restricted to single anatomical structures. Stroke, trauma and tumour do not respect functional anatomical boundaries.

Modern imaging techniques such as CT and MRI have dramatically improved the demonstration of the extent of brain damage caused by many pathologies. Of course, such techniques do not establish the full extent of

anatomical pathology, and even more importantly do not show the distant functional effects (diaschisis) of such lesions.

Nevertheless, the consequences of lesions identified by CT or MRI give some clues as to function. The introduction of CT and MRI has produced many reports of clinicopathological correlation based upon single cases or small series of patients with lesions in particular brain structures.

Deep middle cerebral artery infarction is common and its neurologic picture is dominated by the consequences of internal capsule involvement. Although pure internal capsule infarction has been well documented, infarction limited to the basal ganglia is poorly recognized. Two clinical syndromes associated with lesions limited to the basal ganglia have been defined: (1) behavioral and cognitive disorders associated with infarcts within the caudate nucleus and (2) motor disorders (dystonia) and cognitive disorders associated with disorders in the lentiform nucleus.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Infarction limited only to the basal ganglia is rare. Several studies have been conducted so far to analyze the manifestations of basal ganglionic infarcts. In most of these studies, movement disorders were a selection criterion. But, in our study, case selection depended on the localization of infarction in the basal ganglia without involvement of other structures.

Understanding the organization and physiology of basal ganglia is essential to analyze the manifestations of basal ganglia infarcts.

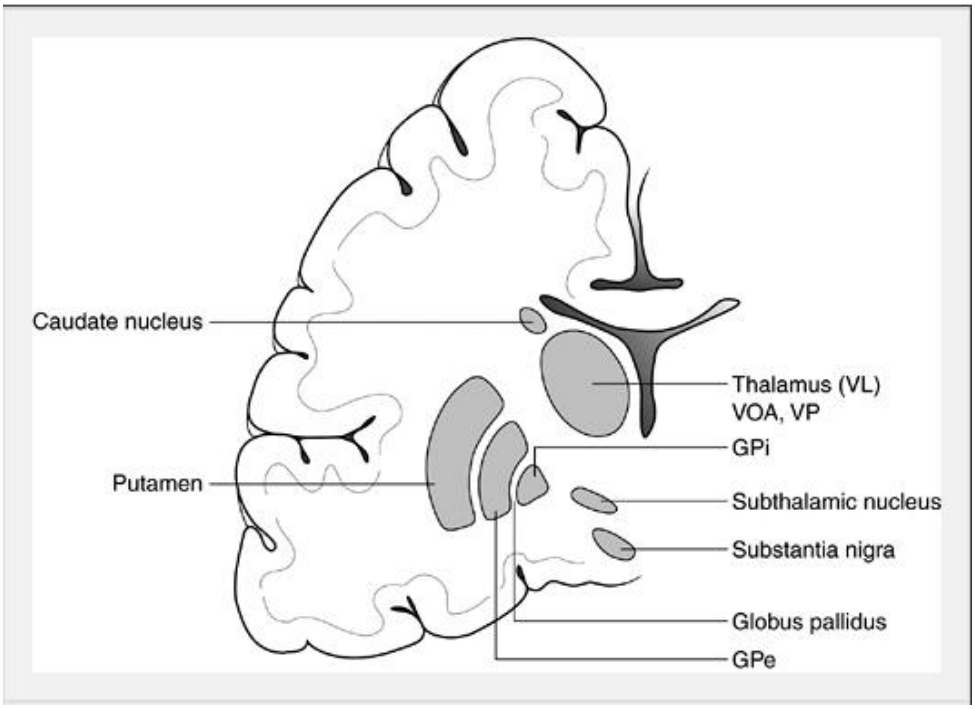
ORGANIZATION AND CIRCUITS OF BASAL GANGLIA:

Basal ganglia include the corpus striatum, the substantia nigra (pars compacta and a pars reticularis), the subthalamic nucleus of Luys, and the ventral tegmental area. The corpus striatum comprises the striatum proper (or neostriatum), made up of the putamen, caudate nucleus, and nucleus accumbens, and the globus pallidus (or paleostriatum), with its medial or internal (Gpi) and lateral or external (Gpe) segments and the ventral pallidum, with its internal and external portions[1].

The major anatomic connections of the basal ganglia are complex and include several closed circuits of connections. In essence, the basal ganglia consist of an input zone, comprising the putamen, caudate nucleus, and ventral striatum, and an output zone, comprising the medial globus pallidus and the

substantia nigra pars reticularis. The main outputs from the medial globus pallidus and substantia nigra pars reticulata are to the thalamus, and thence to the premotor (e.g., supplementary motor area, anterior cingulate motor area, and lateral premotor cortex) and frontal lobe structures.

ANATOMY OF BASAL GANGLIA:



INPUTS INTO THE STRIATUM:

All parts of the cerebral cortex give rise to efferent fibers to the caudate and putamen. These corticostriate projections terminate mainly ipsilaterally in a topographic pattern (e.g., the frontal cortex projects fibers to the ventral head of the caudate and rostral putamen). The cortex also sends fibers to the substantia nigra, subthalamic nucleus, and claustrum. The intra laminar nuclei of thalamus, substantia nigra pars compacta and brainstem raphe nucleus projects

into the striatum. Most striatal efferents project to the globus pallidus. Other striatal efferents go to substantia nigra.

PALLIDAL AFFERENTS AND EFFERENTS:

The globus pallidus receives ascending afferent fibers from the substantia nigra and subthalamus (mainly to the medial or internal pallidum). Both the external and internal globus pallidus also receive afferents from the striatum.

The major outflow from the globus pallidus arises from the internal portion and projects to the ventral anterior (VA) and ventral lateral (VL) nuclei of the thalamus. These thalamic nuclei also receive afferents from the pars reticularis of the substantia nigra. Because the VL thalamic nucleus projects to the motor cortex and the VA thalamic nucleus projects to the premotor cortex, the major basal ganglia efferents influence the motor system.

Efferents from the internal globus pallidus also project to the CM thalamic nuclei, which in turn project to the putamen. The external portion of the pallidum sends fibers to the internal pallidum and to the subthalamic nucleus. The subthalamic nucleus in turn sends fibers to both the internal and external pallidum. Other pallidal efferents also project to the substantia nigra, red nucleus, inferior olive, hypothalamus, and mesencephalic reticular formation.

NIGRAL AFFERENTS AND EFFERENTS:

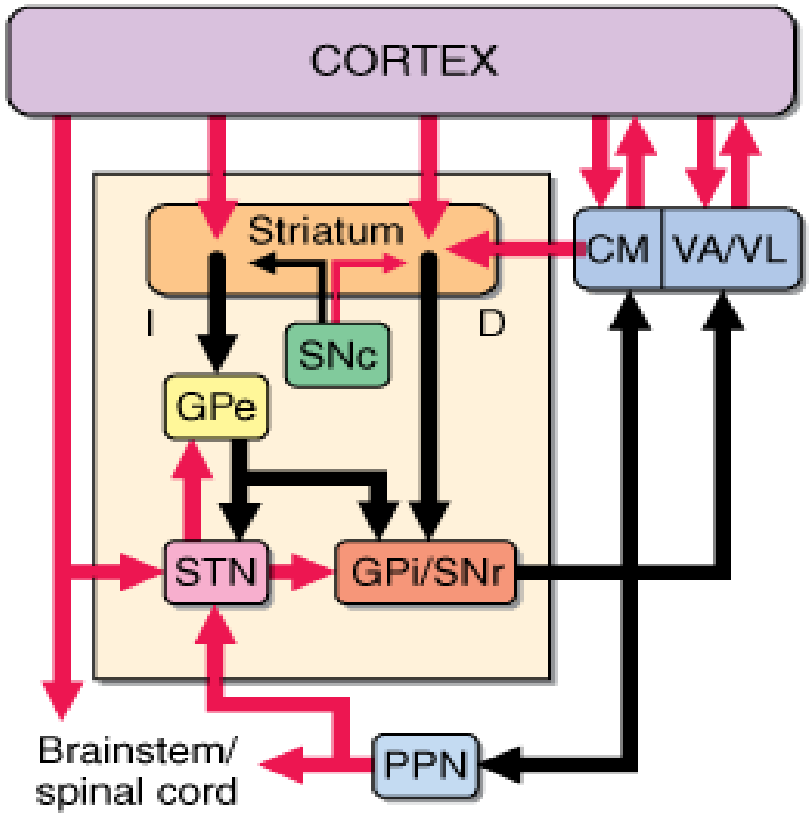
The pars reticularis of the substantia nigra receives fibers from the cerebral cortex, the striatum, the globus pallidus, and the subthalamic nucleus. Pars reticularis efferents project to the VA and VL thalamic nuclei and to the reticular formation and superior colliculus. The pars compacta of the substantia nigra sends dopaminergic fibers to the caudate nucleus and putamen. This output is excitatory for the striatal neurons of the direct pathway and inhibitory to the striatal neurons of the indirect pathway.

It can thus be seen that the basal ganglia exert their influence mainly by way of the cerebral cortex (i.e., they do not send fibers that connect directly with brainstem and spinal cord structures). They provide a subcortical network by which the entire cerebral cortex can influence the motor system (motor and premotor cortex.

Both anatomically and physiologically, a direct and an indirect system have been described in the striato-pallido-thalamic projection. In the direct system, the putamen and the caudate receive excitatory input from the pars compacta of the substantia nigra and project inhibitory fibers to the medial globus pallidus and to the pars reticularis of the substantia nigra which, in turn, inhibit the ventrolateral nucleus of the thalamus. Stimulation of this system would disinhibit the ventrolateral nucleus of the thalamus, resulting in cortical excitation.

In the indirect system, the putamen and caudate receive inhibitory input from the pars compacta of the substantia nigra and project inhibitory fibers to the lateral globus pallidus which, in turn, inhibits (through GABA) the subthalamic nucleus. The subthalamic nucleus stimulates (through glutamate) the medial globus pallidus, inhibitory over the ventrolateral nucleus of the thalamus. Stimulation of this system inhibits the ventrolateral nucleus of the thalamus and results in cortical inhibition.

DIRECT AND INDIRECT PATHWAY IN BASAL GANGLIA



LESIONS OF BASAL GANGLIA:

Pathologic processes affecting the basal ganglia are often diffuse. When discrete, they usually also affect neighboring structures, such as the internal capsule, the hypothalamus, and the white matter of the cerebral hemispheres. Therefore, except for hemiballismus often associated with damage to the contralateral subthalamic nucleus, correlation between basal ganglia lesions and clinical motor dysfunction tends to be obscure.

The literature concerning behavioral effects of lesions of the basal ganglia in experimental animals is often conflicting, and these lesions rarely produce models of human movement disorders. In general, stimulation and destructive lesions of the caudate, putamen, and globus pallidus produce inhibition of movement or contralateral body turning.

Lesions of the subthalamic nucleus produce contralateral hemiballismus. Small unilateral lesions of the anteroventral portion of the caudate cause contralateral choreoathetosis. Unilateral lesions of the globus pallidus may cause contralateral hemidystonia, hemiparkinsonism, or tremor, whereas bilateral globus pallidus lesions may cause dystonia, parkinsonism, abulia, or akinesia.

Lesions of the substantia nigra result in parkinsonism. Unilateral basal ganglia (pallidal-putaminal) hemorrhages or lacunar infarcts may present with sudden falling to the contralateral side while sitting, standing, or walking. The

falls are distinctly slow, tilting motions in a stereotyped lateral or diagonal trajectory and occur with the eyes open but are exacerbated by eye closure.

In a study[2] of behavioral and movement disorders with lesions affecting the basal ganglia, lesions of the caudate nucleus rarely caused motor disorders (e.g., chorea or dystonia) but were more likely to cause behavioral problems, especially abulia (apathy with loss of initiative and of spontaneous thought and emotional responses) or disinhibition. Lesions of the putamen and globus pallidus rarely caused abulia and did not produce disinhibition but commonly caused dystonia, particularly when the putamen was involved.

Bilateral lesions of either the putamen or the globus pallidus caused parkinsonism or dystonia-parkinsonism infrequently. The prominence of behavioral disturbances with caudate lesions emphasizes the more complex cognitive role of this structure, whereas the frequent occurrence of dystonia and less commonly parkinsonism with lesions of the putamen and globus pallidus emphasizes the motor roles of these structures.

In another study[4] of patients with lenticular infarcts, two distinct clinical syndromes corresponding to the two anatomical areas of the lenticular nucleus were described. Infarcts within the globus pallidus were associated with behavioral and cognitive disorders, whereas infarcts in the putamen caused motor disorders (dystonia) and cognitive impairment. Also, damage to a frontal-caudate functional system likely underlies the aphasias (language

disturbances) resulting from subcortical lesions affecting the deep frontal and paraventricular white matter (subcortical aphasias).

Movement disorders can be defined as neurologic dysfunctions in which there is either an excess of movement (Abnormal Involuntary Movements, or AIMs; hyperkinesias; dyskinesias) or a paucity of voluntary and automatic movements (akinesia, bradykinesia, or hypokinesia) unassociated with weakness or spasticity. Paucity of movement characterizes the disorder known as parkinsonism.

STUDIES ON BASAL GANGLIA INFARCTS:

Khailash P Bhatia and C.David Marsden analyzed the behavior and movement disorders in basal ganglionic lesions[2]. The behavioral and movement disorders reported in 240 patients described in the literature with lesions affecting the caudate nucleus, putamen and the globus pallidus (lentiform nucleus) have been analyzed. Amongst the 240 cases, dystonia was the most frequent movement disorder recorded (36%); chorea (8%) and parkinsonism (6%) or dystonia-parkinsonism (3%) were uncommon.

The commonest behavioral disturbance was the syndrome of abulia (apathy with loss of initiative and of spontaneous thought and emotional responses) (13%); disinhibition was rare (4%). Chorea has been described in only 6% of those with caudate lesions, and dystonia in only 9%. The most significant behavioral disturbance described in 28% of those with caudate

lesions was the syndrome of abulia, sometimes alternating with disinhibition (11%).

Lesions of the lentiform nuclei rarely caused abulia (10%) and did not produce disinhibition, but they commonly caused dystonia (49%), particularly when the putamen was involved (63%). Bilateral lesions of the lentiform nuclei, either of the globus pallidus or of the putamen, caused parkinsonism (19%) or dystonia- parkinsonism (6%) infrequently.

The prominence of the behavioral disturbance of abulia with caudate lesions emphasizes the more complex cognitive role of this basal ganglia structure. The frequent occurrence of dystonia and less commonly of parkinsonism with lentiform lesions emphasize the motor roles of putamen and globus pallidus.

In this study, the following conclusions were drawn after the analysis of cases. (i)Lesions of the caudate nucleus, even unilateral, may cause abulia, or more rarely disinhibited behavior. (ii) Lesions of the caudate nucleus infrequently cause motor disorders. If they do, it usually is chorea or dystonia, and almost never parkinsonism. (iii) Lesions of the lentiform nucleus (putamen and globus pallidus) infrequently cause abulia or disinhibition, and if they do the globus pallidus is usually involved. (iv) Lesions of the lentiform nucleus commonly cause dystonia, and rarely cause chorea. Lesions involving the putamen are more prone to cause dystonia than those involving the globus

pallidus. (v) Bilateral lesions of the lentiform nucleus, usually involving the globus pallidus, infrequently cause parkinsonism or dystonia-parkinsonism.

Emre Kumral et al evaluated the clinical profile of acute caudate vascular lesions[3]. 31 patients were analyzed. Caudate infarct was present in 25 patients and caudate hemorrhage in 6. The most frequent neurological abnormalities were abulia and psychic akinesia (48%), frontal system abnormalities (26%), speech deficits in patients with left-sided lesions (23%), and neglect syndromes in those with right-sided lesions (10%). Patients with infarct in the territory of the lateral lenticulostriate arteries extending to neighboring structures showed more frequent motor and neuropsychological deficits than those with infarct in the territory of the anterior lenticulostriate arteries.

Heike Russman et al analyzed the clinical features and topographic correlation of acute lentiform infarct in 13 patients[4]. All had faciobrachiocrural hemisyndrome, while none showed acute or delayed parkinsonism or abnormal movement. Nine patients had a lesion restricted to the putamen. Two of them had ataxic motor hemisyndrome and 7 had sensorimotor hemisyndrome (with ataxia in 4, left hemineglect in 1, and deep pain in the arm and leg in 1).

Four patients had a lesion of putamen and globus pallidus externus. Three of them had motor hemisyndrome (with nonfluent aphasia in 2 and ataxia in 1) and 1 had ataxic sensorimotor hemisyndrome. All infarcts were in

the territory of the medial perforating branches of the medial cerebral artery. Presumed cause of stroke was small-artery disease in 5, artery-to-artery embolism in 4, cardioembolism in 3 and undetermined in 1. Movement disorders were not described.

M Giroud et al analyzed the clinical features[5] of unilateral lenticular infarct in 20 patients. Two distinct clinical syndromes were identified corresponding to the two anatomical areas of the lenticular nucleus: behavioral and cognitive disorders were associated with infarcts within the globus pallidus, whereas both motor disorders (dystonia) and cognitive disorders were associated with infarcts within the putamen.

Outcome was excellent in all the patients for motor function, but slight cognitive disorders, problems with short term memory, and dysphasia persisted for several months. The size of the lesion did not explain these symptoms. The author proposed that slight reduction in cerebral blood flow found in the adjacent frontotemporal area[6] may explain them by a deafferentation or a diaschisis phenomenon.

Lesions involving the lentiform nucleus, particularly the putamen, caudate nucleus, thalami, and parietal cortex, often in combination, have been associated with acute and subacute posthemiplegic focal dystonia or hemidystonia[7]. However, there was extension of the lesion into adjacent structures, including the internal capsule, in most of the cases.

Other motor disorders, such as unilateral chorea, hemichorea-hemiballism, asterixis, acute stereotypies, acute focal dystonia[8], and subacute parkinsonism, have also been reported after unilateral lesions of the lentiform nucleus but often also involve the caudate nucleus or the internal capsule. Lesions involving the globus pallidus may cause behavioral and speech disorders[9], as well as motor disorders including delayed contralateral hemidystonia or subacute choreoathetosis. In most cases, there is a delay of months to years between the onset of the actual lesion[10] and the development of the motor disorders.

Hemichorea-hemiballismus[11] secondary to lacunar infarct in the basal ganglia region is rare. Goldblatt et al[12] described a patient with what was called hemichorea in whom lesions were found to be restricted to the contralateral head of the caudate nucleus and putamen at autopsy, and Kase et al[13] described a 54-year-old man with hemichorea-hemiballismus in whom CT scan revealed a lacunar infarct in the contralateral putamen and caudate nucleus. It has therefore been suggested that the syndrome of hemichorea-hemiballismus should be included in the group of clinical syndromes that are most commonly caused by lacunar infarcts.

The pathogenesis of hemichorea-hemiballismus secondary to a lacunar infarct in the basal ganglia is not well understood. The choreoballistic movements in this patient could have been caused by destruction of the caudate nucleus and putamen, thereby reducing the inhibitory outflow from these

structures to the globus pallidus and substantia nigra[14]. On the other hand, ischaemia may have stimulated neurons that were left intact, activating dopamine production and release and thus reducing the inhibitory influences of the substantia nigra and globus pallidus on cortical motor function.

The pathologic process responsible for the dystonia during the initial recovery period is unknown. Some authors have compared the interval of delayed-onset dystonia in patients of perinatal anoxia and stroke. They emphasized the age of hypoxic injury in deciding the duration of delay in spite of the differences of etiology.

Delayed onset dystonia is a rare sequelae of stroke. The anatomical basis and pathogenesis of delayed-onset dystonia is uncertain. Mitchell suggested that the delay in the onset of hemichorea or athetosis following hemiplegia was caused by progressive changes in the original brain lesion. Burke (1980)[15] had hypothesized that the mechanism of delayed-onset dystonia occurring a year or more after the insult may be due to aberrant neuronal sprouting.

Pettrigrew and Jankovic (1985)[16] studied 22 patients of delayed onset dystonia following variable causes. They found the mean interval in the seven patients who had brain lesions below the age of 7 was much longer than the other patients, and postulated that the age of the patient at the time of cerebral injury influenced the latency of dystonia from the acute brain damage.

However, the hypothesis of Pettrigrew has limitations. Some patients with immediate onset could not be explained with neuronal regeneration.

Young Chul Chai et al[17] analyzed 34 patients with cerebro vascular disease in the literature with delayed onset dystonia. Dystonia following stroke almost always appeared within 1-12 months (mean 6.5 months). There were four patients with contralateral basal ganglia lesions, who had a short delay despite the early age of hypoxic insult.

One patient with dystonia following stroke in young age had a shorter interval compared to other elderly patients with dystonia following stroke. The pathological lesions in the patient of delayed-onset dystonia following stroke have a variety of anatomical lesion sites.

Of 34 patients reported in the literature, 21 (61.7%) had basal ganglia lesion, 13 (38%) internal capsule lesion, 11 (32%) thalamic lesion, 2 (5.8 %) cortical lesion and 2 (5.8%) without any lesions. Damage to the neuronal circuit connecting caudate, putamen, globus pallidus and thalamus seems to be responsible for the dystonia following stroke. However, it can occur without radiological evidence of striatal lesion. Thalamic degeneration following striatal lesion or cortical lesion has also been reported**.**

The most commonly involved arterial territories in caudate ischemic stroke were the territory of the lateral lenticulostriate arteries from the middle cerebral artery[18] and the territory of the anterior lenticulostriate arteries from

the anterior cerebral artery. In previous studies, there is no mention of arterial territories involved in patients with caudate infarcts. Actually, there is considerable overlap between the 3 arteries supplying the head of the caudate nucleus: the lateral lenticulostriate, anterior lenticulostriate, and Heubner's recurrent artery.

Anterior lenticulostriate arteries primarily circumscribed the MCN, LCN, and VCN and partially involved the anterior limb of the capsule. Infarctions in the territory of the lateral lenticulostriate arteries were limited to the MCN, LCN, VCN, anterior part of the internal capsule, and putamen[19]. From a clinical standpoint, infarcts in the territory of the anterior lenticulostriate arteries yield only mild neuropsychological deficits, while those with infarcts in the territory of the lateral lenticulostriate arteries presented prominent motor and neuropsychological deficits.

The most prominent clinical features of basal ganglionic vascular lesions were behavioral and cognitive abnormalities, as in previous studies. Behavioral changes may have occurred as a result of loss of function in cortical zones, caused by loss of striatal efferent projections from the caudate nucleus. The caudate nucleus is the principal crossing area of basal ganglia–thalamo cortical loops.

As defined by different authors, the caudate nucleus connects associative cortex, including frontal, parietal, and temporal lobes, with deeper

anatomic structures by cortico-pallido-nigra-thalamocortical loops[20]. These loops are multiple, discrete, but partially overlapping and are integrated through their passage in pallidum and substantia nigra to the circumscribed nuclei of the thalamus, and from there they are projected back to their original lobar areas.

In a study by Emre Kumral et al on acute caudate vascular lesions in 31 patients, one half of the patients had abulia, characterized by decreased spontaneous activity and speech and prolonged latency in responding to questions and other stimuli. Three patients had psychic akinesia, characterized by severe mental and affective stagnation and lack of initiative for action and speech. Trillet et al observed psychic akinesia in 3 patients with apathy, flattened affect, lack of initiative for usual daily activities, stereotyped behaviors, and prolonged akinetic attacks. Moreover, these features were previously reported in patients with bilateral globus pallidus or putaminal lesions.

The abulic patients described by Fisher[21] had lesions in the frontal lobes and underlying structures or in the thalamus and upper brain stem. Caplan et al[22] described 10 abulic patients with left-sided preponderance of caudate lesions. Among them, 4 patients had isolated caudate lesions, others had involvement of the anterior limb, and only 1 had spread to the putamen.

The mechanism of abulia was explained by interruption of the limbofrontal connection[23]. The quantitative and temporal features of behavior were more difficult to measure, and a variety of descriptive terms have been used such as bradykinesia, abulia, psychic akinesia, and akinetic mutism. These terms describe a continuum from minor to major absence of observable behavior, and despite the advanced behavioral stage, some intellectual and cognitive functions could be retained.

AIMS AND OBJECTIVES

OBJECTIVES AND AIMS OF THE STUDY

1. To evaluate the motor features of basal ganglia infarcts such as the hyperkinetic and hypokinetic movement disorders.

2. To evaluate the cognitive impairment and it’s severity in basal ganglia infarcts.

3. To evaluate the behavioral and mood disturbances in basal ganglia infarcts.

MATERIALS AND METHODS

MATERIALS AND METHODS

STUDY DESIGN AND PERIOD:

Our study was a prospective observational study, conducted in Government General Hospital, Chennai, from January 2009 to March 2011. Patients were selected from Neurology OP, emergency ward, general medical wards and Neurology ward. Patients were properly informed about the study and the Institutional Ethics Committee clearance was obtained. We studied all the patients with basal ganglia infarcts either acute or chronic with the following eligibility criteria. Patients were diagnosed with the aid of CT or MRI. Patients who were not affordable for MRI scan were subjected only to CT study of brain.

INCLUSION CRITERIA:

1. Patients who were admitted in Medical and Neurological wards for acute ischemic stroke with CT or MRI evidence of acute or chronic basal ganglia (putamen, caudate, globus pallidus or subthalamic nucleus) infarcts with or without infarcts in the internal capsule.

2. Patients who attended Neurology OPD, or admitted in Neurology and Medical wards with CT or MRI evidence of chronic basal ganglia infarcts with or without infarcts in the internal capsule.

EXCLUSION CRITERIA:

1. Patients with CT or MRI evidence of infarcts in a site other than basal ganglia and internal capsule.

2. Patients with past history of intracerebral hemorrhage.

3. Past history of head injury or encephalitis.

4. Previous history of psychiatric, mood or personality disorder and longstanding antipsychotic drug intake.

5. Evidence of coexisting neurodegenerative dementia or epilepsy.

6. Presence of chronic medical illness like heart failure, chronic kidney disease, cirrhosis of liver and hypothyroidism.

7. Evidence of systemic connective tissue disorder like, SLE and Rheumatoid arthritis

8. History of alcohol abuse or other illicit drug abuse.

CLINICAL ASSESSMENT:

All the patients selected for the study underwent detailed neurological examination including higher mental functions, motor, sensory, cranial nerve and cerebellar functions. Patients were carefully assessed for the presence of hypokinetic or hyperkinetic movement disorder. Rapid alternating movement of fingers, finger tapping, foot tapping and gait assessment was done to diagnose bradykinesia. Patients were carefully examined for the presence of rigidity, dystonia, choreo athetotic movements.

Detailed higher mental function assessment was done. Attention span, language function (fluency, repetition, comprehension, naming, reading, and writing) and memory function (immediate, recent and remote memory) were assessed.

Frontal lobar functions were assessed by trail making test, Luria's motor series, rhythmic tapping test, stroop test, alternate sequence tasks, and assessment of judgment, abstract thinking and reasoning power.

Parietal lobar functions were assessed by evaluation of cortical sensations (tactile localization, two point discrimination, stereognosis, and graphesthesia), constructional ability, dressing ability, visuospatial orientation, right left orientation, calculation and ideomotor apraxia.

Temporal lobar functions were assessed by detailed verbal, visual memory examination, paired associate learning and story recalling ability. Occipital lobar functions were assessed by analysis of gnosis for familiar objects and faces.

With detailed history taking, patients were probed for the presence of behavioral, mood and sleep disturbances. Standard rating scales and questionnaire were used to assess the mood and behavior disturbance. Beck Depression Inventory II and Hamilton Anxiety Scale were used to assess the depression and anxiety respectively. Probing questions were asked to ascertain whether the patient had behavioral disturbances like delusions, hallucinations, irritability, apathy and disinhibition. Patients were assessed for the presence of behavior disorder by Neuropsychiatric Inventory (NPI) and scoring was given.

After the detailed higher mental function examination, patient's cognitive function was scored with Mini Mental Scale Examination and Addenbrooke's cognitive scoring.

INVESTIGATIONS:

All the patients underwent routine biochemical investigations like blood sugar, urea, creatinine, lipid profile, complete hemogram, liver function tests, blood VDRL, HIV serology. CT brain was performed for all the patients using horizontal orbitomeatal sequences with 0.5mm sections.

MRI brain was performed for the patients who can afford to pay the cost of MRI imaging. Those who cannot pay for the MRI imaging underwent only CT imaging of brain. MRI brain was performed on a 1.5 Tesla MRI machine with conventional sagittal T1, axial T2 and FLAIR sequences. TOF MR Angiogram was also performed during MRI procedure.

All the patients underwent four vessel Doppler studies. ECG, X-ray chest PA view and echocardiogram was done for all the patients to search for the cardiac source of embolism like mural thrombus, wall hypokinesia or akinesia, aneurysm and atrial fibrillation

On CT, recent infarct was defined as hypodense grey lesion without dilatation of ipsilateral ventricle. Chronic infarct was defined as hypodense lesion of CSF density with dilatation of ipsilateral ventricle. Lacunar infarct was defined as infarcts of size less than 1.5cm with sharply defined areas of hypodensity which may be round, rectangular or pyramidal. Infarcts of more than 1.5cm were defined as large infarcts.

Small artery disease was considered in the presence of only lacunar infarcts with diameter less than 1.5mm, hypertension, diabetes, absence of large vessel disease in Doppler study and absence of cardiac source of embolism by echocardiography.

Large artery disease was considered in the presence of stenosis of more than 50% of appropriate large artery on Doppler study and larger size of the infarct (>1.5cm).

Hypertension, diabetes mellitus, hypercholesterolemia, smoking and alcoholism were considered as risk factors.

RESULTS

RESULTS

51 patients fulfilled the eligible inclusion and exclusion criteria and were analyzed in this study.

SEX:

Among the 51 patients who were included in the study, 35 patients were males and 16 patients were females.

AGE:

The average age of disease presentation in this study was 54 years. The youngest patient was a 14 year old girl and eldest patient was 71 year old male. Most of the patients were in the range of 51 to 60 years. The following table shows the age distribution of the patients.

TABLE 1: AGE DISTRIBUTION OF PATIENTS:

AGE IN YEARS	NUMBER OF PATIENTS	PERCENTAGE
61 – 70	8	15.%
51 – 60	26	51%
41 – 50	15	29.4%
31 – 40	1	2%
14 YEARS	1	2%

CEREBROVASCULAR RISK FACTORS:

Among 51 patients in this study, 36 patients were hypertensive, 23 patients were diabetics, 14 patients were hypertensive and diabetic, 10 patients had hyperlipidemia and 10 patients had past history of stroke. 30 patients were smokers and 24 were alcoholics.

TABLE 2: CEREBROVASCULAR RISK FACTORS:

RISK FACTORS	NUMBER OF PATIENTS	PERCENTAGE
HYPERTENSION	36	70.59
DIABETES MELLITUS	23	45.1
HYPERTENSION AND DIABETES	14	27.45
HYPERLIPEDEMIA	10	19.61
PAST H/O STROKE	10	19.61
SMOKING	30	58.82
ALCOHOLISM	24	47.06

SOURCE OF BASAL GANGLIA INFARCTS:

Among 51 patients, 20 patients had basal ganglionic infarcts due to presumed large vessel disease. These 20 patients had larger size of the infarct (>1.5cm in diameter). Of these 20 patients, 17 patients had evidence of large

vessel occlusion in the ipsilateral carotid artery. The remaining 3 patients had cardiac source of embolism; 2 patients (48 and 40 years) suffered from rheumatic heart disease with severe mitral stenosis and atrial fibrillation; 1 patient (50 year old male, diabetic) suffered from ischemic heart disease with evidence of left ventricular mural thrombus in transthoracic echocardiogram.

30 patients had basal ganglionic infarcts due to presumed small vessel disease. These patients had lacunar infarcts in the unilateral or bilateral basal ganglia with infarct diameter less than 1.5cm and had normal carotid and vertebral Doppler study and echocardiogram. Of these 30 patients, 1 patient (42 year old male) had left basal ganglionic and capsular infarct due to small vessel vasculitis. He had evidence of systemic vasculitis in the form of bilateral retinal vasculitis, vasculitic leg ulcers, elevated ESR and young onset systemic hypertension.

A 14 year old girl who was admitted with acute right hemiparesis and dysarthria had acute infarct in the left globus pallidum and internal capsule. Detailed young stroke work up was done, but no source of infarct was revealed. She suffered from migraine with visual aura and developed stroke during the peak of one headache episode. She also had past history suggestive of transient global amnesia.

TABLE 3: SOURCE OF BASAL GANGLIA INFARCTS:

SOURCE	NUMBER OF PATIENTS	PERCENTAGE
LARGE ARTERY DISEASE	17	33.33
SMALL ARTERY DISEASE	30	58.82
CARDIAC SOURCE	3	5.88
UNKNOWN	1	1.97

PATIENT CHARACTERISTICS:

After selection into the study, the 51 patients were analyzed with detailed clinical assessment and laboratory investigations including neuro imaging. They were categorized into 4 groups according to their presenting symptomatology.

GROUP I:

Patients who presented with acute neurological deficit due to ischemic stroke with CT or MRI evidence of acute infarct involving the basal ganglia with or without internal capsular infarct were included in this group. They presented with hemiparesis, movement disorder or behavioral disturbance. They had at least one symptom or sign referable to basal ganglia infarct.

This group comprised of 18 patients. Of these 18 patients, 8 patients presented with hemiparesis; Among the 8 patients, 3 patients had motor aphasia, 2 patients had hemidystonia. These 8 patients in addition had signs of behavioral and cognitive impairment on examination. 7 patients presented with acute onset of hemichorea or choreoathetosis. 2 patients presented acutely with hemiballismus. 1 patient (60 year old male) presented with acute onset dementia, in the form of disinhibited behavior, memory impairment, visuospatial disorientation and emotional incontinence.

TABLE 4: GROUP I:

PRESENTING FEATURE	NUMBER OF PATIENT
HEMIPARESIS ONLY	3
L HEMIPARESIS WITH APHASIA	3
HEMIPARESIS WITH DYSTONIA	2
HEMICHOREA	7
HEMIBALLISMUS	2
DEMENTIA	1

GROUP II:

Patients who presented with longstanding symptoms (non acutely) in the form of movement disorder, behavioral or cognitive disorders with imaging evidence of chronic basal ganglia infarcts were included in Group II. On

detailed clinical assessment, all their symptoms and signs were referable to basal ganglia infarcts.

This group comprised of 14 patients. Of 14 patients, 7 patients had dementia as the major clinical feature. Of these 7 patients, 2 patients in addition had parkinsonian features. In 2 patients, bilaterally symmetrical parkinsonism was the dominant clinical feature. These 2 patients had also cognitive impairment. In 5 patients, dystonia was the major clinical feature. Of 5 patients, 1 patient had parkinsonian feature and 2 patients had cognitive impairment.

TABLE 5: GROUP II:

MAJOR FEATURE	NUMBER OF PATIENTS
DEMENTIA	7
PARKINSONISM	2
DYSTONIA	5

GROUP III:

Patients who had basal ganglia infarcts in neuro imaging, but had no symptoms or signs referable to basal ganglia infarcts were included in Group III. This group comprised of 13 patients. These patients were again classified into 2 sub groups.

IIIA: Patients, who presented to the Neurology OPD for some other non specific complaints like headache and giddiness, underwent neuro imaging and imaging showed basal ganglia infarcts were included in this group. On detailed clinical assessment, they had no symptoms or signs referable to basal ganglia infarcts. This group consisted of 5 patients.

IIIB: Patients who were admitted with acute onset hemiparesis with imaging evidence of basal ganglionic and capsular infarcts were included in this group. They also had no symptoms or signs referable to basal ganglia infarcts. This group consisted of 8 patients.

TABLE 6: GROUP III:

GROUP III	**PATIENT FEATURE**	**NUMBER OF PATIENTS**
GROUP IIIA	AYMPTOMATIC BASAL GANGLIONIC INFARCTS	5
GROUP IIIB	ACUTE HEMIPARESIS WITH ASYMTOMATIC BASAL GANGIONIC INFARCTS	8

GROUP IV:

Patients, who presented to the Neurology OPD with some other non specific symptoms, underwent neuro imaging and imaging showed basal ganglionic infarcts were included in this group. On detailed clinical assessment, they had some signs referable to basal ganglionic infarcts, either cognitive impairment or motor feature. They were not aware of their cognitive or motor impairment.

This group comprised of 6 patients. All these patients had evidence of cognitive impairment and mood disorder on detailed clinical assessment. 1patient (55 year old male) in addition had right hand dystonia.

CLINICAL FEATURES:

MOTOR FEATURES:

Of 51 patients, 22 patients had motor features related to basal ganglia infarct. Of these 22 patients, 7 patients had hemichorea, 2 patients had hemiballismus, 8 patients had dystonia, 2 patients had parkinsonism and 3 patients had parkinsonism with dystonia. All the 7 patients with chorea and 2 patients with hemiballismus presented acutely to the emergency ward (group 1). All 5 patients with parkinsonism presented non acutely to the Neurology OPD (group II) with bilaterally symmetric akinetic rigid syndrome.

Of 11 patients with dystonia, 2 patients presented acutely with dystonia as a manifestation of acute ischemic stroke. Of these 2 patients, 1 patient had

hand dystonia along with hemiparesis and another patient had hemidystonia with hemiparesis. For 8 patients, dystonia confined predominantly to the unilateral hand; 2 patients had hemidystonia and 1 patient had blepharospasm. 2 patients developed dystonia in the limbs which suffered hemiparesis several years back; they had chronic infarct in the contralateral putamen, pallidum and capsule; they were diagnosed as post hemiplegic dystonia.

All patients with hemichorea had contralateral infarct in the caudate nucleus. 2 patients with hemiballismus had contralateral infarct in the sub thalamic nucleus. All 5 patients with parkinsonism had the evidence of bilateral basal ganglia infarcts; 2 patients had bilateral pallidal infarct and 3 patient had bilateral infarcts in the lentiform nucleus; among these 5 patients, 2 patients had bilateral caudate infarct and 2 patients had unilateral caudate infarct. Of 11 patients with dystonia, all had either unilateral or bilateral putaminal or pallidal infarct and 4 patients had caudate infarct.

TABLE 7: MOTOR FEATURES:

MOTOR FEATURES	NUMBER OF PATIENTS	PERCENTAGE
HEMICHOREA	7	31.82
HEMIBALLISMUS	2	9.09
DYSTONIA	8	36.36
PARKINSONISM	2	9.09
PARKINSONISM WITH DYSTONIA	3	13.64

COGNITIVE FEATURES:

Of 51 patients, 27 patients had evidence of cognitive impairment on detailed higher mental function examination. Decreased attention span, executive dysfunction and impaired verbal memory recall were the common cognitive problems in these patients. Some of the patients also had language disturbance, visuospatial disorientation and calculation difficulty.

Of 27 patients with cognitive impairment, 11 patients belonged to Group I (acute presentation). Among these 11 patients, cognitive impairment was the presenting feature in one patient; in all other patients motor impairment was the presenting feature and they had cognitive impairment on examination. 10 patients belonged to Group II (non acute presentation); and 6 patients belonged to Group IV.

Decreased attention span was the most common abnormality noted; 24 patients had evidence of reduced attention span on digit repetition test, go no go test, letter cancellation test and trail making test. 17 patients had evidence of executive dysfunction on alternate sequence tasks, trail making, stroop test, Luria's motor series and rhythmic tapping test. 19 patients had memory impairment on verbal memory recall (improving with cues), paired associate learning, story recall and tests of orientation to time and place.

3 patients had language disturbance. These 3 patients presented acutely (Group I) with left hemiparesis and aphasia. The aphasia was expressive

aphasia with reduced word fluency, word finding difficulty, grammatical errors, impaired repetition, naming, reading, writing and preserved comprehension. These 3 patients had acute large infarct involving left caudate, putamen, pallidum and capsule.

10 patients had difficulty in performing complex calculation (oral and wrote); 2 patients had visuospatial disorientation with way finding difficulty, difficulty in identifying cities in the map and defective visuospatial tasks on Addenbrooke's cognitive assessment; 4 patients had constructional disability in copying diagrams.

Cognitive functions were scored with Mini Mental Scale Examination (MMSE) and Addenbrooke's cognitive scoring. Of 27 patients with cognitive impairment, MMSE scale was in the range of 16 to 27 and Addenbrooke's cognitive scoring was in the range of 61 to 97.

Patients with unilateral basal ganglionic infarct had less severe cognitive impairment and better scoring in Addenbrooke's scoring (85 – 97). Patients with bilateral basal ganglionic infarcts had more severe cognitive impairment and poor scoring (61- 85). Patients with unilateral or bilateral caudate infarcts had more severe and more frequent cognitive impairment than isolated lentiform infarcts. Patients with combined caudate and lentiform infarcts had more frequent cognitive impairment than isolated lentiform infarcts. All patients with calculation difficulty had left caudate infarct.

TABLE 8: COGNITIVE FEATURES:

COGNITIVE IMPAIRMENT	NUMBER OF PATIENTS	PERCENTAGE
POOR ATTENTION SPAN	24	47.06
EXECUTIVE DYSFUNCTION	17	33.33
MEMORY IMPAIRMENT	19	37.25
APHASIA	3	5.8
CALCULATION ERRORS	10	19.61
VISUOSPATIAL DISORIENTATION	2	3.92
CONSTRUCTIONAL DISABILITY	4	7.84

BEHAVIORAL FEATURES:

Of 51 patients, 29 patients had behavioral abnormality in the form of apathy, loss of mental and motor drive, depression, anxiety, disinhibition, irritability, delusion, hallucination or sleep disturbance. Apathy was the most common abnormality noted. Of 27 patients, 25 patients had apathy and loss of drive. Depression was the next common abnormality noticed. When the patients were analyzed with Beck's Depression Inventory II, 27 patients had evidence of depression. All these patients showed the features of sadness, pessimism, loss of pleasure, feeling of worthlessness and loss of interest. No

patients had suicidal ideas. Of 27 patients, 4 patients had severe depression with score more than 29. 9 patients had moderate depression with score 20 to 29. 8 patients had mild depression with score between14 to 20. 6 patients had mild depression with score less than 14.

8 patients had features of anxiety as assessed by Hamilton's Anxiety Scale. 7 patients had disinhibited behavior in the form of unconcerned micturition, undressing in front of others and abusing bad words. 4 patients had delusions and 4 patients had formed visual hallucinations. 22 patients had evidence of sleep disturbance. The most common sleep disturbance was insomnia with delayed sleep onset. 11 patients had irritability and recurrent outburst of anger.

All these patients were scored with Neuropsychiatric Inventory Questionnaire. The highest score was 44 and the lowest score was 8. Patients with bilateral basal ganglionic infarct and with severe cognitive impairment had higher scoring and severe behavioral abnormality. Patients with infarcts in the caudate nucleus (unilateral or bilateral) had more frequent behavioral abnormality than patients with isolated lentiform nucleus infarcts.

TABLE 9: BEHAVIORAL FEATURES:

BEHAVIORAL ABNORMALITY	NUMBER OF PATIENTS	PERCENTAGE
APATHY	25	49.02
DEPRESSION	27	52.94
ANXIETY	8	15.69
DISINHIBITION	7	13.73
DELUSION	4	7.84
HALLUCINATION	4	7.84
INSOMNIA	22	43.14
IRRITABILITY	11	21.57

INFARCT CHARACTERISTICS:

Of 51 patients, 18 patients underwent MRI imaging of the brain and 33 patients underwent CT imaging of the brain.16 patients had bilateral basal ganglionic infarcts; 35 patients had unilateral basal ganglionic infarcts. 28 patients had associated internal capsular infarct, of which 1 patient had bilateral capsular infarct. 36 patients had infarct in the caudate nucleus; 25 patients had unilateral caudate infarct; 11 patients had bilateral caudate infarct. 17 patients had infarct in the putamen; 16 patients had unilateral putamen infarct and 1 patient had bilateral putamen infarct. 25 patients had infarct in the globus pallidus; 20 patients had unilateral pallidal infarct and 5 patients had bilateral pallidal infarct. 2 patients had infarct in the subthalamic nucleus.

Of 51 patients, 25 patients had infarcts confining only to the caudate nucleus (either unilateral or bilateral). 13 patients had isolated lentiform infarcts (either unilateral or bilateral putamen or pallidum). 11 patients had infarcts in both caudate and lentiform nucleus. 2 patients had infarct in the unilateral subthalamic nucleus.

Of 51 patients, 16 patients had larger size of the infarct occupying more than one basal ganglionic structure and internal capsule with infarct size greater than 1.5cm. 35 patients had smaller size of the infarcts with infarct diameter less than 1.5cm.

TABLE 10: INFARCT CHARACTERISTICS:

INFARCT LOCATION	NUMBER OF PATIENTS	PERCENTAGE
UNILATERAL CAUDATE	25	49.02
BILATERAL CAUDATE	11	21.57
UNILATERAL PUTAMEN	16	31.37
BILATERAL PUTAMEN	1	1.96
UNILAT PALLIDUM	20	39.22
BILATERAL PALLIDUM	5	9.80
SUBTHALAMIC NUCLEUS	2	3.92
INTERNAL CAPSULE	28	54.90

TABLE 11: DISTRIBUTION OF INFARCTS:

INFARCTS	NUMBER OF PATIENTS	PERCENTAGE
ISOLATED LENTIFORM INFARCTS	13	25.49
ISOLATED CAUDATE INFARCT	25	49.02
CAUDATE AND LENTIFORM INFARCTS	11	21.57
SUBTHALAMIC NUCLEI INFARCT	2	3.92

CORRELATION OF IMAGING AND CLINICAL FEATURES:

TABLE 11: MOTOR DISORDERS PRODUCED BY BASAL GANGLIONIC INFARCTS:

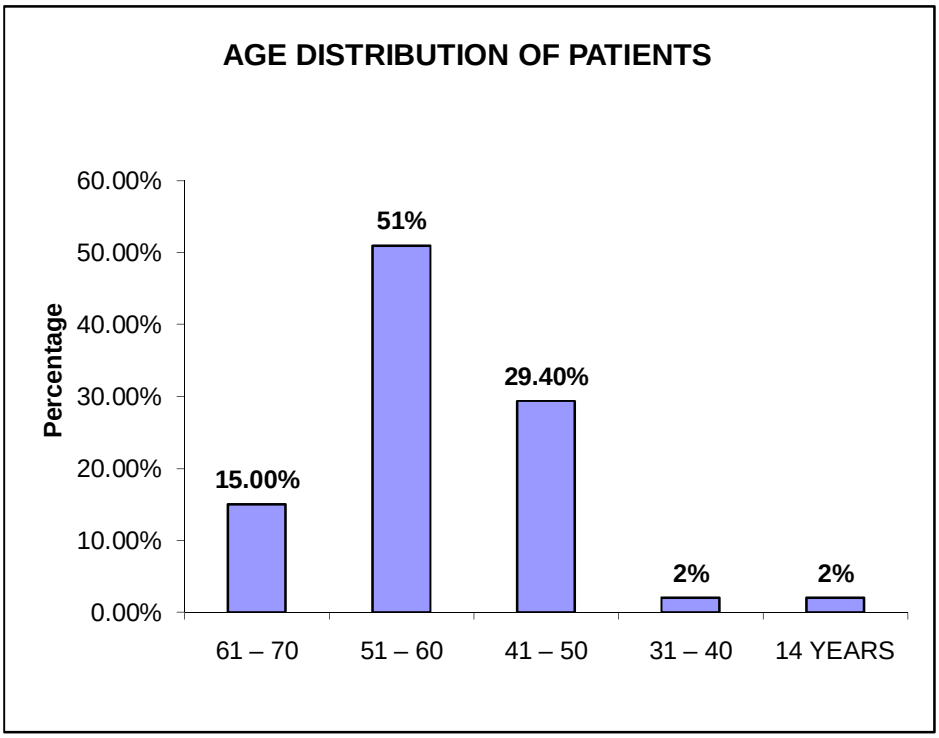
	Caudate Infarct		Putamen Infarct		Pallidal infarct		Lenti form	Caudate And Lenti form	Sub Thalamic	Total
	UNI	BI	UNI	BI	UNI	BI				
Hemichorea	6	--	--	--	--	--	--	1	--	7
Hemiballismus	--	--	--	--	--	--	--	--	2	2
Dystonia	--	--	--	--	--	--	6	2	--	8
Parkinsonism	--	--	--	--	--	--	--	2	--	2
Dystonia with parkinsonism	--	--	--	--	--	--	1	2	--	3

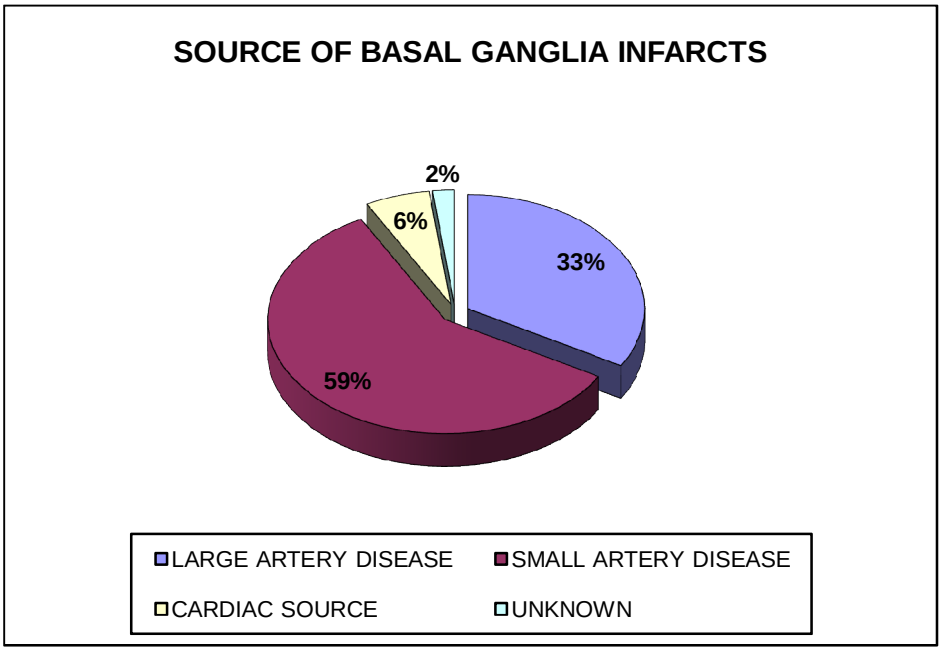
TABLE 12: COGNITIVE IMPAIRMENT PRODUCED BY BASAL GANGLIA INFARCTS:

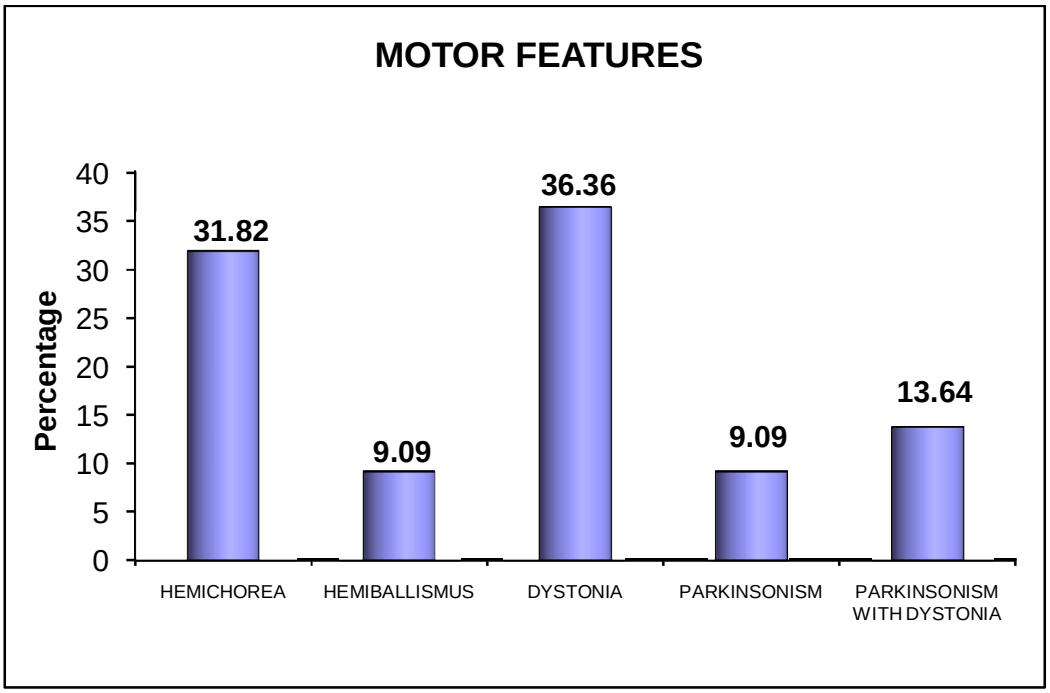
	Caudate Infarct		Putamen Infarct		Pallidal Infarct		Lenti form	Caudate and lentiform	Total
	UNI	BI	UNI	BI	UNI	BI			
POOR ATTENTION SPAN	7	6	--	--	--	--	3	8	24
EXECUTIVE DYSFUNCTION	2	6	--	--	--	--	1	8	17
MEMORY IMPAIRMENT	7	5	--	--	--	--	1	6	19
APHASIA	--	--	--	--	--	--	--	3	3
CALCULATION ERRORS	3	4	--	--	--	--	1	2	10
VISUOSPATIAL DISORIENTATION	--	1	--	--	--	--	--	1	2
CONSTRUCTIONAL DISABILITY	1	--	--	--	--	--	--	3	4

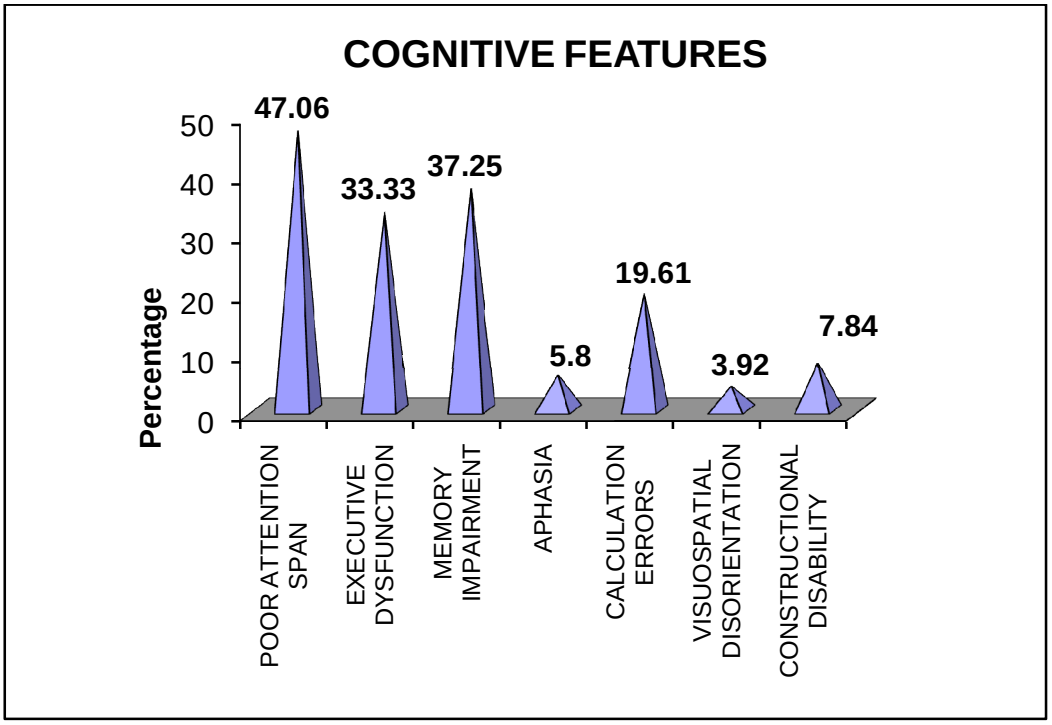
TABLE 13: BEHAVIORAL DISORDER PRODUCED BY BASAL GANGLIA INFARCTS:

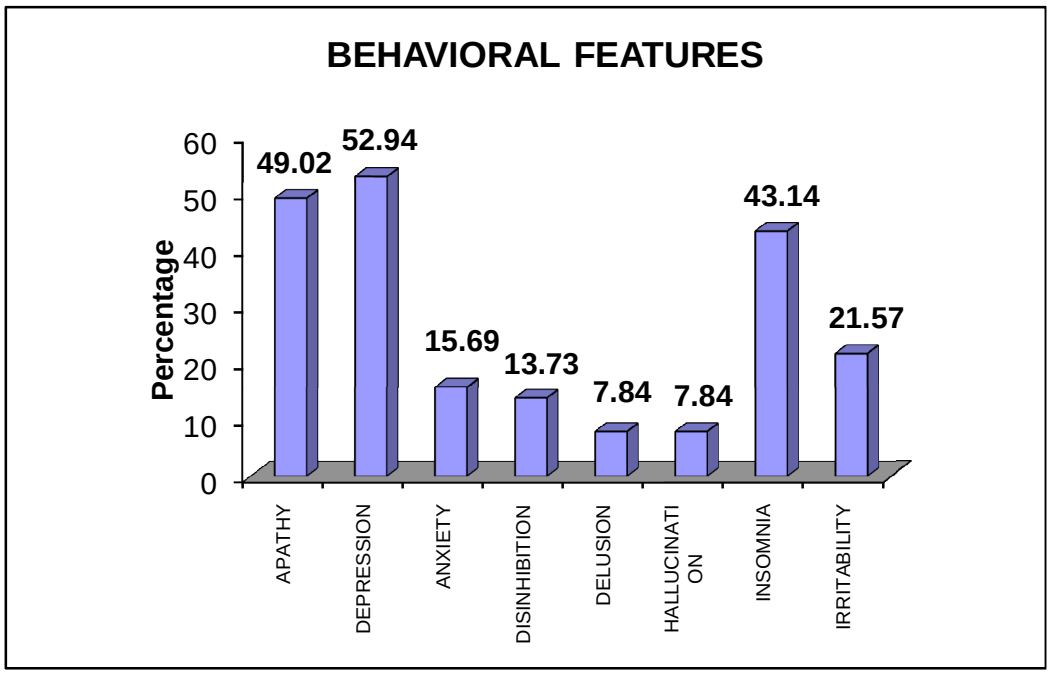
	Caudate Infarct		Putamen Infarct		Pallidal Infarct		Lenti Form	Caudate And Lenti-form	Sub Thal-amic	Total
	UNI	BI	UNI	BI	UNI	BI				
APATHY	6	7	--	--	--	--	3	9	--	25
DEPRESSION	7	7	--	--	--	--	2	10	1	27
ANXIETY	1	1	--	--	--	--	2	4	--	8
DISINHIBITION	--	4	--	--	--	--	--	3	--	7
DELUSION	--	2	--	--	--	--	--	2	--	4
HALLUCINATION	--	2	--	--	--	--	--	2	--	4
INSOMNIA	6	6	--	--	--	--	3	6	1	22
IRRITABILITY	3	4	--	--	--	--	2	2	--	11

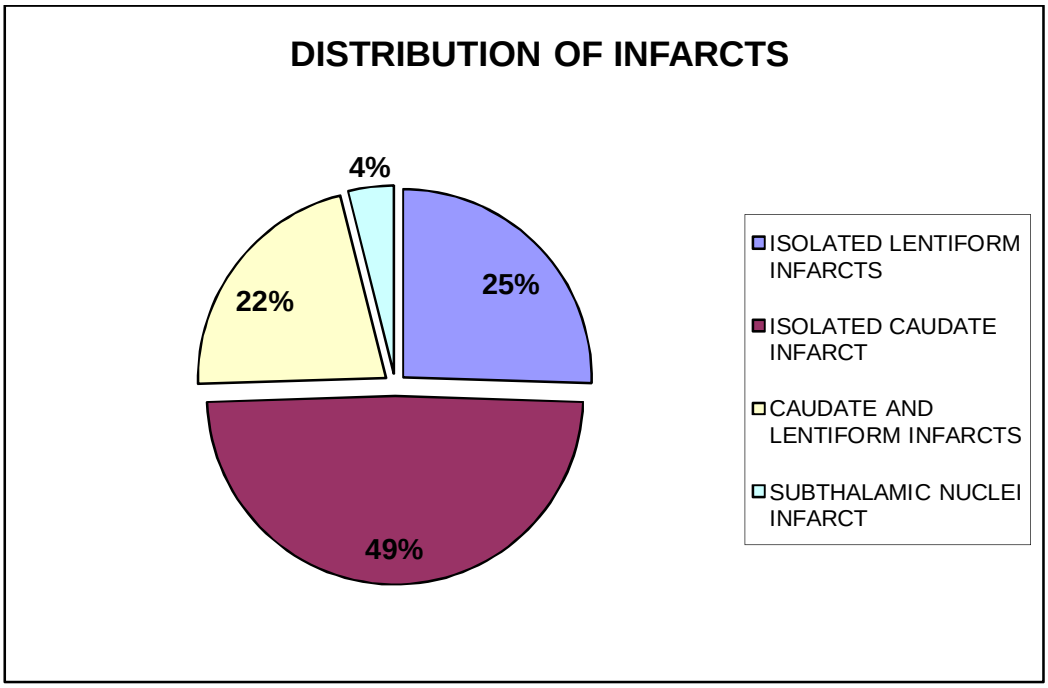

AGE DISTRIBUTION OF PATIENTS
Percentage
60.00%
50.00%
40.00%
30.00%
20.00%
10.00%
0.00%
15.00%
51%
29.40%
2%
2%
61 – 70
51 – 60
41 – 50
31 – 40
14 YEARS


SOURCE OF BASAL GANGLIA INFARCTS
2%
6%
33%
59%
LARGE ARTERY DISEASE
SMALL ARTERY DISEASE
CARDIAC SOURCE
UNKNOWN


MOTOR FEATURES
Percentage
40
35
30
25
20
15
10
5
0
31.82
9.09
36.36
9.09
13.64
HEMICHOREA
HEMIBALLISMUS
DYSTONIA
PARKINSONISM
PARKINSONISM WITH DYSTONIA


COGNITIVE FEATURES
Percentage
50
40
30
20
10
0
47.06
33.33
37.25
5.8
19.61
3.92
7.84
POOR ATTENTION SPAN
EXECUTIVE DYSFUNCTION
MEMORY IMPAIRMENT
APHASIA
CALCULATION ERRORS
VISUOSPATIAL DISORIENTATION
CONSTRUCTIONAL DISABILITY


BEHAVIORAL FEATURES
Percentage
60
50
40
30
20
10
0
49.02
52.94
15.69
13.73
7.84
7.84
43.14
21.57
APATHY
DEPRESSION
ANXIETY
DISINHIBITION
DELUSION
HALLUCINATION
INSOMNIA
IRRITABILITY


DISTRIBUTION OF INFARCTS
4%
25%
22%
49%
ISOLATED LENTIFORM INFARCTS
ISOLATED CAUDATE INFARCT
CAUDATE AND LENTIFORM INFARCTS
SUBTHALAMIC NUCLEI INFARCT

CASE 16 : INFARCT IN LEFT CAUDATE AND LENTIFORM NUCLEUS IN A PATIENT WITH R HEMIPARESIS AND MOTOR APHASIA

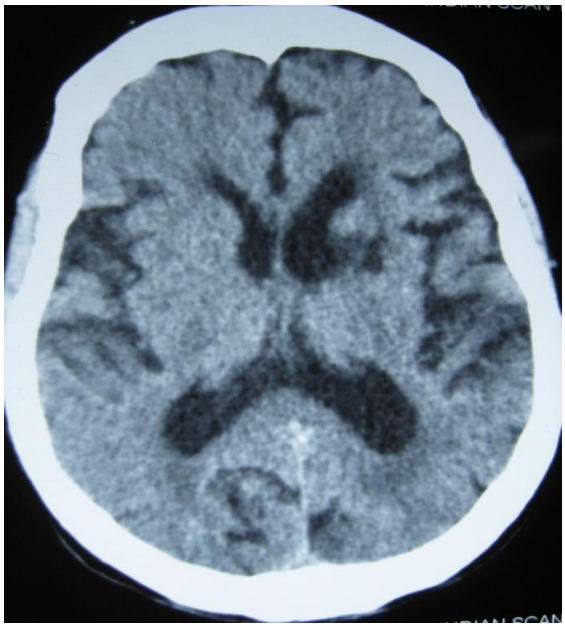


Fig. 1

CASE 12 : ACUTE INFARCT IN R SUBTHALAMIC NUCLEUS IN A PATIENT WITH LEFT HEMIBALLISMUS

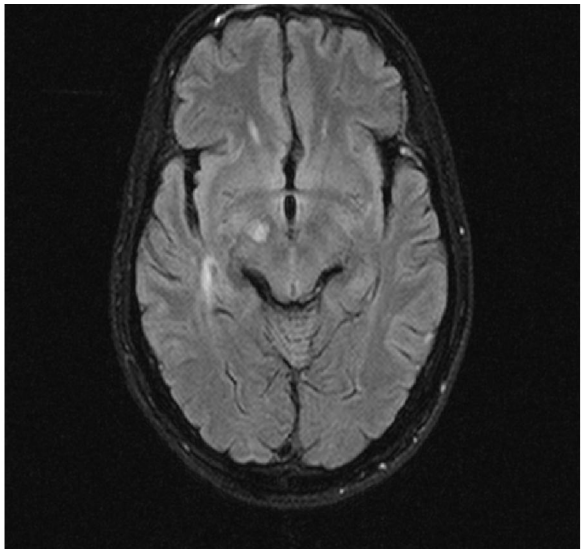


Fig . 2

CASE 1: BILATERAL CAUDATE AND PALLIDAL INFARCT

Fig.3

CASE 21: POST HEMIPLEGIC DYSTONIA
RIGHT CAUDATE, LENTIFORM INFARCT

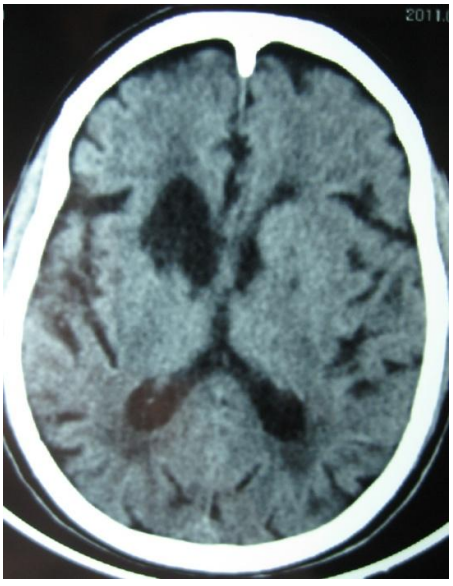


Fig. 4

CASE 5: ACUTE INFARCT LEFT PALLIDUM AND PUTAMEN WITH ACUTE RIGHT HEMIPARESIS

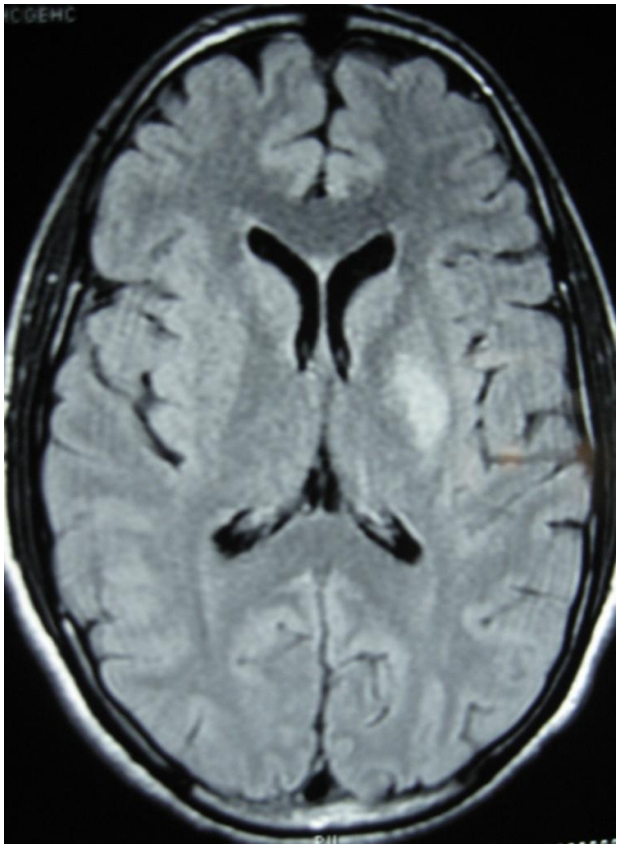


Fig. 5

DISCUSSION

DISCUSSION

Pure basal ganglionic infarcts are rare. Often the acute ischemic infarct extends into the adjacent internal capsule or white matter structures, resulting in neurological deficit referable to the internal capsule. The profound hemiparesis and dysarthria often dominate the clinical picture and mask the neurological impairment produced by basal ganglia infarcts. The cognitive and behavioral abnormalities of basal ganglia infarcts are often overlooked and may disable the activities of daily living. In our study, we analyzed the motor, cognitive and behavioral abnormalities of basal ganglia infarcts.

MOTOR FEATURES:

In our study, among 51 patients with basal ganglionic infarcts, 28 patients had infarcts extending to the adjacent internal capsule (acute and chronic). 18 patients had hemiparesis on clinical examination either as an acute presenting manifestation or as a longstanding residual deficit due to past stroke. As hemiparesis is not directly related to the basal ganglia lesion, we analyzed motor features other than hemiparesis to assess the impact of basal ganglia infarct.

DYSTONIA:

Dystonia was the most common movement disorder observed. 11 patients had dystonia (21.6%) in this study. 9 patients had focal dystonia

involving one hand, 2 patients had hemidystonia and 1 patient had blepharospasm. 2 patients had acute dystonia. 2 patients had delayed dystonia several years after the insult. Hemidystonia was rare in our study, only 2 patients had hemidystonia. Generalized dystonia was not observed in our patients, although several patients had bilateral caudate or lentiform infarcts.

Among 11 patients with dystonia, 7 patients (63.6%) had isolated lentiform nucleus infarct with; 6 patients had unilateral and 1 patient had bilateral lentiform infarct. 4 patients had combined caudate and lentiform infarcts. No patients with isolated caudate infarct developed dystonia. Infarcts in the lentiform nucleus were more frequently associated with dystonia.

Though exact mechanism of dystonia is little known, it has been proved in various studies on basal ganglionic lesions that most lesions causing dystonia were in the lentiform nucleus, particularly the putamen[24]. It seems that crude destruction of the putamen is more likely to cause dystonia than any other form of movement disorder. Such crude putaminal lesions might be expected to destroy both the direct striato-pallidal (nigral) pathway supporting cortically generated movement, and the indirect pathway via lateral globus pallidus and subthalamus which eventually inhibits thalamo cortical excitation of premotor cortical areas[25].

Kailash P.Bhatia and C.David Marsden, in their meta analysis of basal ganglionic lesions found that dystonia was the most common movement

disorder observed in basal ganglionic lesions. M.Giroud et al, in his study on unilateral lentiform infarcts, reported that dystonia occurred in 63% of the putaminal lesion and in 37% of the globus pallidus lesions. In this study, most lesions inducing dystonia were in the lentiform nucleus, mostly when putamen was involved. In our study, patients with dystonia had combined involvement of both putamen and globus pallidus.

2 patients in our study developed dystonia several years after the initial neurological insult. These 2 patients had chronic infarct in the contralateral lentiform nucleus and internal capsule. They had past history of hemiparesis several years (10 and 7 years) ago. Suggested pathophysiological mechanisms for delayed dystonia are aberrant neuronal sprouting, ephaptic transmission after the insult, remyelination and late inflammatory changes[26].

CHOREA:

Next to dystonia, chorea was the common movement disorder. 7 patients had chorea in this study (13.7%). All patients had chorea involving one side limbs (hemichorea). 6 patients had isolated infarct involving the contralateral caudate nucleus. 1 patient had large infarct involving contralateral caudate, lentiform nucleus and internal capsule and also had mild hemiparesis in addition to hemichorea.

This finding is in contrast to the results of Meta analysis by Khilash Bhatia et al. In his analysis, chorea was a rare movement disorder, occurred in

only 18 of the 240 patients (8%); most patients with chorea (15 of 18; 83%) had involvement of the caudate nucleus. Only three of 68 (4%) lesions affecting the lentiform nucleus, but sparing caudate, were associated with chorea.

Striatal projections to the globus pallidum externum are probably more severely involved in patients with chorea and basal ganglionic lesion. Injection of GABA antagonists into the lateral pallidum, which block striatal inhibition of the lateral globus pallidus, can provoke contralateral hemichorea in monkeys (Crossman *et al*, 1988)[27]. 2-Deoxyglucose activity was greatly increased in the subthalamus indicating increased lateral pallidal inhibition of this nucleus (Mitchell *et al.,* 1985)[28]. This was associated with 2-DG evidence of decreased subthalamomedial pallidal excitation and reduced pallido-thalamic inhibition[29].

Hemichorea or hemiballism can be produced reliably by lesions of the subthalamus or subthalamo-pallidal pathways in primates and man[30] (Martin, 1957; Whittier and Mettler,

1949; Carpenter et al.1950)[31]. This appears to be due to loss of subthalamic excitation of the medial pallidum, with disinhibition of pallido-thalamic targets[32].

PARKINSONISM:

In our study, 5 patients had parkinsonism (9.8%). All these 5 patients had bilaterally symmetric parkinsonism with akinetic rigid phenotype. All of them belonged to group II (chronic presentation). No patients in the acute presentation group (group I) had parkinsonism. No patients had hemi parkinsonism. In these 5 patients, bradykinesia involved the upper and lower limbs equally without any preferential distribution to the lower limbs.

Interestingly, 1 patient (case 52) had akinetic rigid syndrome, eyelid stare, vertical gaze paresis and frequent backward falls; the initial working diagnosis was progressive supranuclear palsy. But, subsequently MRI brain revealed right caudate and bilateral pallidal infarct. His parkinsonian features improved well with levo dopa.

All the 5 patients had bilateral basal ganglionic infarcts; 4 patients had bilateral pallidal and caudate infarcts and 1 patient had bilateral putaminal and unilateral pallidal infarct.

In 3 patients (case no 15, 33, 52), parkinsonism was the predominant presenting feature. In the remaining 2 patients, dementia was the predominant presenting feature and in addition they had parkinsonism. 2 patients had isolated parkinsonism and 3 patients had dystonia in addition to parkinsonism (2 with hand dystonia, 1 with blepharospasm).

Our findings are comparable with the Meta analysis of Khailash Bhatia and Marsden. In their analysis, parkinsonism also was relatively uncommon, being seen in only 21 of the 240 patients (9%). In 14 cases, the parkinsonism was relatively pure, but seven others had additional dystonic features. Nineteen of these 21 parkinsonian patients had lesions involving the lentiform nuclei.

Striatal lesions causing loss of both its excitatory effect on the direct striato pallidal pathway, and its inhibitory effect on the indirect pathway, thus withdrawing striato pallido thalamo cortical support of cortically generated movement, could explain the parkinsonism[33]. Lesions of the human globus pallidus cause parkinsonism infrequently, and have to be bilateral to do so[34]. In our study, 2 patients with parkinsonism had bilateral pallidal infarct without involvement of putamen, but they had associated caudate infarct.

BEHAVIORAL FEATURES:

33 patients in this study (64.7%) had one or more behavioral abnormality. Of these only 12 patients (or their relatives) had volunteered that they had behavioral abnormality. In all other patients, behavioral abnormality was found only with detailed neuropsychiatric assessment. This indicates that behavioral abnormality in basal ganglionic infarcts is like the tip of iceberg. Many patients with basal ganglionic infarcts may suffer from behavioral disorder without clinical attention and treatment.

APATHY:

Apathy was the most common behavioral abnormality noticed. 25 patients (49%) in this study had apathy with lack of mental and motor drive. The severity of apathy was assessed with NPI (Neuropsychiatric Inventory). Apathy was seen both in acute and chronic basal ganglionic infarcts. Most of these patients had infarcts in the caudate nucleus. 6 patients had unilateral and 7 patients had bilateral caudate infarcts. 9 patients had combined caudate and lentiform infarct; 3 patients had isolated lentiform infarcts.

Most of the patients (88%) with apathy had caudate infarcts. Apathy (12%) was rare in isolated lentiform infarcts. In Meta analysis by Khailash Bhatia, the commonest behavioral disorder was abulia, which manifested in 30 of the 240 (13%) cases. In 23 of these 30 (77%) patients with abulia, the lesion involved the caudate nucleus.

The loss of mental and motor initiative and drive that characterizes abulia, causing apathy and blunting of responses, does seem to be most commonly due to damage to the caudate and sometimes to the globus pallidus, which may normally be involved in these functions[35].

In contrast to apathy, disinhibited behavior occurred only in 7 patients (13.7%). Disinhibited behavior was in the form unconcerned micturition, undressing and urinating in the public places and violent behavior. All these patients had caudate infarcts; 4 patients had bilateral isolated caudate infarcts; 2

had bilateral caudate with lentiform infarct and one had unilateral caudate with lentiform infarct. In contrast to apathy which occurred in both unilateral and bilateral caudate lesions, disinhibited behavior was rare in unilateral caudate lesions and occurred predominantly in bilateral caudate lesions.

Abulia, and less frequently disinhibition, does seem to be a significant consequence of caudate damage. The massive projection from prefrontal cerebral cortex to the head of the caudate nucleus and the reciprocal striato-pallido thalamo- cortical projections back to prefrontal cortex via the 'complex' basal ganglia circuits (Alexander *et al.,* 1986, 1990)[36] emphasize the strength of prefrontal-caudate systems. Indeed abulia and disinhibition are recognized clinical consequences of damage to prefrontal cortex in man. Both behavioral syndromes can therefore be produced by damage either to prefrontal cortex or to the caudate nucleus[37].

Apathy is considered to be due to damage to the anterior cingulate frontal basal ganglionic circuit[38]. The anterior cingulate gyrus projects to the ventral striatum, thence to ventral and rostrolateral globus pallidus and rostrodorsal substantia nigra, the paramedian medial dorsal thalamic nucleus, which projects back to the anterior cingulate cortex.

Disinhibited behavior[39] is considered to be due to damage to the lateral orbito frontal circuitry. The lateral orbitofrontal cortex projects to the ventromedial caudate nucleus, thence to the dorsomedial pallidum and

rostromedial substantia nigra, medial ventral anterior and dorsal thalamic nuclei, which project back to the orbito-frontal cortex[40].

Depression was observed in 27 patients (53%). But, only 12 patients had severe depression on Beck's Depression Inventory. Most of these patients had sadness, pessimistic attitude, loss of pleasure, self dislike, loss of interest in household and occupational activities, worthless feeling and crying. No patients had suicidal ideas. Of these 27 patients, no patients had volunteered their depressive symptoms. All these patients presented to the hospital only because of their motor disability or other behavioral impairment.

14 patients with depression had isolated caudate infarcts, 7 had unilateral and 7 had bilateral caudate infarct. 10 patients had caudate and lentiform infarct. Only 2 patients with isolated lentiform infarct had depression.

Reiko Sato et al[41] studied the correlation of depression with MRI evidence of lesion in the basal ganglia and non basal ganglia region. In his study, depression scale scores were not independently associated with basal ganglia lesions. Other factors like age, gender, socio economic status and past history of stroke also contributed to the development of depression. K.R.Ramakrishnan et al[42], in his study on depression in elderly individuals, found that patients with basal ganglionic lesions are more prone to develop depression, especially those who had caudate lesions.

4 patients (7.8%) had delusion and 4 patients (7.8%) had hallucination. In all 4 patients, the delusion was in the form of suspecting the life partner whether she had illegal contact with some other person. 4 patients had formed visual hallucination in the form of viewing close relatives or grandparents though nobody actually existed. No patients had auditory hallucinations. All these patients with delusion and hallucination had severe dementia with cognitive impairment.

All 4 patients with delusion had bilateral caudate infarct, 2 patients in addition had lentiform infarct. Among 4 patients with visual hallucinations, 2 patients had isolated caudate infarct and 2 patients had caudate and lentiform infarct.

McMurtray et al[43] in his study on patients with lacunar infarction in the caudate nucleus, found that patients with caudate infarcts and delusions had greater frontal-executive impairments on neuropsychological measures and decreased metabolism of the inferior prefrontal cortex on PET imaging. These findings support the hypothesis that development of delusions was consequent to disruption of inferior prefrontal lobe functions, which may include alterations in reality monitoring and testing.

11 patients (21.56%) had irritability and recurrent outburst of anger for trivial problems. Of these, 7 patients had isolated caudate infarct, 2 patients had isolated lentiform infarcts and 2 patients had caudate and lentiform infarcts. All

the patients with behavioral and mood disorder had caudate infarcts. Isolated lentiform infarcts rarely produced behavioral or mood disorder. This shows the importance of frontal basal ganglionic circuitry in producing behavioral abnormality.

This study shows that behavioral and mood disorders are very common in patients with basal ganglionic infarcts. All the patients with basal ganglionic infarcts should be thoroughly explored for the presence of behavioral disorder. Identification and appropriate treatment of behavioral disorder[44] and depression in these patients will reduce the morbidity and will definitely improve the quality of life.

COGNITIVE FEATURES:

Of 27 patients with cognitive impairment, only 10 patients had cognitive impairment as their presenting feature; remaining patients presented with motor features or some other non specific complaints, but cognitive impairment was identified on detailed examination. So, cognitive impairment in basal ganglionic infarct is more than what we see, again like the tip of iceberg (like behavioral abnormality).

POOR ATTENTION SPAN:

Poor attention span was the most common cognitive abnormality noticed. 24 patients (47%) had poor attention span. They performed poorly on

attentional tasks like digit repetition test, go no go test, serial subtraction test, letter cancellation test and trail making test.

13 patients had isolated caudate infarcts, 7 had unilateral and 6 had bilateral infarcts. 8 patients had combined caudate and lentiform infarcts. Only 3 patients had isolated lentiform infarcts.

Though poor attention span has no specific localizing value, brain regions involved in attentional mechanisms can be separated into two categories: activated sensory areas and brain structures that activate them. The brain regions activated are inferior parietal lobule, premotor areas, cingulate gyrus and posterior temporo occipital areas. Activation of these cortical areas requires input from the basal ganglia through the thalamus. Several studies have shown that basal ganglionic lesions can cause poor attention span[45].

EXECUTIVE DYSFUNCTION:

\17 patients (33.33%) had executive dysfunction as evidenced by alternate sequence tasks, trail making, stroop test, Luria's motor series and rhythmic tapping test. Of these, 8 patients had isolated caudate infarcts, 2 with unilateral and 6 with bilateral infarcts. 8 patients had caudate and lentiform infarcts. 1 patient alone had isolated lentiform infarct.

Executive dysfunction was probably due to involvement of dorsolateral prefrontal subcortical circuitry which projects to the caudate nucleus[46].

The dorsolateral prefrontal syndrome is characterized by defects of executive function and motor programming. Such patients are unable to generate hypotheses and cannot maintain or shift set. They also exhibit reduced verbal and design fluency, with defective strategic learning and motor execution of alternating or sequential tasks[47]. The dorsolateral prefrontal cortex projects primarily to the dorsolateral head of caudate nucleus, thence to dorsomedial globus pallidus and rostral substantia nigra, ventral anterior and medial dorsal thalamic nuclei, which project back to the dorsolateral prefrontal region[48].

MEMORY IMPAIRMENT:

19 (38%) patients had evidence of memory impairment and defective new learning tasks. Of these, 12 patients had isolated caudate infarcts, 7 with unilateral and 5 with bilateral infarcts. 6 patients had caudate and lentiform infarcts. Only 1 patient had isolated lentiform infarct. These patients had impairment on tasks like verbal memory recall, paired associate learning and word category fluency tasks.

Though basal ganglia is not contributing its participation to the neuro anatomical circuitry (Papez circuit) for short term memory (explicit memory), it has been found in various studies that short term memory recall could be impaired in basal ganglia lesions alone[49].

The caudate has been hypothesized to play a distinct role in memory. In monkeys, for example, stimulation of the caudate has been shown to impair memory[50]. Mendez et al reported on 11 patients with unilateral or bilateral caudate infarction and 1 patient with a healed abscess who came to medical attention because of an acute behavioral disturbance[51]. Among these patients, 1 patient with bilateral lesions was judged to have a "global dementia" with an MMSE score of 18. Moreover, among 7 patients with unilateral lesions, there were significant impairments in tasks requiring planning and sequencing compared with age-matched control subjects. They also had short attention spans and decreased free recall of episodic and semantic items.

Caplan et al[22] also reported on the clinical and CT findings in 18 patients with unilateral caudate infarction. Memory impairment was observed in 2 patients who had left-sided lesions, and they were also found to be "abulic and slow." Richfield et al[52] reported a patient with bilateral caudate damage examined at 8 months from onset or 1 year later who had cognitive impairment, especially in delayed recall. Tatemichi et al[53] reported that a combination of psychomotor slowing or abulia and memory impairment was the most striking behavioral feature among stroke patients with dementia. In his study, 6 of 21 patients with caudate lesions showed abulia as determined by a report of lack of drive or motivation in response to a question on the PSE, and 3 of these 6 patients showed both memory impairment and MMSE scores below 20.

Pozzilli et al[54] suggested that the deficit in verbal comprehension and verbal memory may have been due to dysfunction of the cortico caudate connections. On the basis of these prior studies, the cognitive impairment in our patients might be assumed to indicate that caudate lesions lead to chronic deficits in frontal lobe function. This chronic frontal lobe dysfunction gradually leads to dysfunction in other connected cortical regions, and this is manifested by cognitive decline. Other explanations such as chronic dysfunction of the caudate might also be proposed as an explanation for these findings.

Emre Kumra et al[3] studied the behavioral features in acute caudate stroke. Among 31 patients with acute caudate infarct, 3 patients with unilateral caudate infarct had verbal memory impairment on Rey auditory verbal learning test and 3 patients had combined verbal and visual memory impairment. This may be due to disconnection of caudate nucleus from frontal lobe.

APHASIA:

3 patients had language disturbance in the form of motor aphasia. All 3 patients presented acutely with large infarct involving left caudate, lentiform nucleus and internal capsule. These patients had hesitant and interrupted speech with word finding difficulties, with impaired repetition, reading, writing, naming and intact comprehension. They also had literal paraphasia.

Kumra et al[3], in his study on acute caudate lesions on 31 patients, found that 6 patients had aphasia; 4 patients had nonfluent aphasia, 1 patient had

transcortical motor aphasia and 1 patient had global aphasia; 5 patients had left caudate infarct and 1 patient with non fluent aphasia had bilateral caudate infarct.

It is well known that different types of aphasia, such as transcortical, nonfluent aphasia, characterized by semantic and verbal paraphasias and perseverations without comprehension impairment, occur in patients with left caudate or anterior lentiform vascular lesions[55]. Alexander et al observed that patients with subcortical lesions involving the caudate nucleus, anterior limb of the internal capsule, and putamen had word-finding difficulty or hesitancy without severe aphasic abnormalities. It is probable that acute disconnection of linguistic pathways between anterior and posterior speech areas, which are connected with the left caudate nucleus and anterior limb of the internal capsule, may yield a different type of aphasia[56].

Though transcortical aphasia has been described frequently in basal ganglionic lesions, in our study no patient had transcortical aphasia; all had non fluent aphasia (Broca's aphasia).In most of the patients with aphasia due to dominant striatocapsular infarct, the infarct also extends into the adjacent white matter of insula and temporal lobe[57].

Giroud et al[5], in his study on 20 patients with unilateral lentiform infarcts, he noted an acute but regressive dysphasia characterized by hypophonia, verbal paraphasia, non-fluent speech with silence, loss of words

without any impairment of comprehension, and preserved repetition in five right handed patients with left putaminal infarction.

VISUOSPATIAL DISORIENTATION AND CONSTRUCTIONAL DISABILITY:

In our study, 2 patients had visuospatial disorientation and 4 patients had constructional disability. Of 2 patients with visuospatial disorientation, 1 patient had isolated bilateral caudate infarct and 1 patient had caudate and lentiform infarct. Of 4 patients with constructional disability, 1 patient had isolated caudate infarct and 3 had caudate and lentiform infarct.

Hemineglect was occasionally reported in patients with right caudate lesions and has not been systematically studied in either infarct or hemorrhage[58]. In one series, contralateral hemineglect was reported in 3 cases with right caudate lesion involving the anterior limb of the internal capsule. Moreover, the putamen and adjacent white matter lesions are also involved in the development of hemineglect[59], but in this series, the lesions were all large and involved neighboring structures. All these 3 patients had visuospatial disorientation and constructional disability as well. In our study, no patients had hemineglect.

CALCULATION DIFFICULTY:

10 patients in this study had calculation difficulty. Of these, 7 had isolated caudate infarct; 4 had bilateral caudate infarct and 3 had left caudate infarct. 1 patient had isolated lentiform infarct and 2 patients had combined caudate and lentiform infarct.

In a PET study by Stanislas et al[60], multiplication activities yielded superior activity in the left lentiform nucleus, left inferior parietal gyrus and bilateral fusiform and lingual gyri. In his study, he concluded that left basal ganglia would be involved in storing and retrieving rote verbal multiplication facts.

Elina et al[61], in a study compared the patterns of acalculia in two patients, one with a left parietal lesion and the other with a left basal ganglia lesion. The patients were tested on a broad range of neuropsychological abilities, with the main focus on number processing and calculation. The results show many similarities between their deficits, with some difficulties in simple arithmetic, arithmetical rules and mental and written complex calculations. The errors made in complex mental and written calculations were due to memory-based procedural impairments in both patients. These findings, corroborated with other studies reported in the literature, suggest the existence of a fronto-parieto-subcortical circuit responsible for arithmetic complex

calculations and that procedural knowledge relies on a visuo-spatial sketchpad that contains a representation of each sub-step of the procedure.

ASYMPTOMATIC PATIENTS WITH BASAL GANGLIONIC INFARCTS:

Of 51 patients, 13 patients had asymptomatic basal ganglionic infarcts. They had no symptoms or signs referable to basal ganglia. Of 13 patients, 5 patients presented to the Neurology OPD with non specific complaints like head ache or giddiness. CT brain showed basal ganglionic infarcts. 8 patients presented to the Emergency ward with acute hemiparesis. They had acute infarct in the internal capsule and basal ganglia. But, they had no signs or symptoms referable to basal ganglia. All these patients had at least one of the cerebro vascular risk factors like hypertension, diabetes mellitus, hyperlipidemia or cigarette smoking.

Of 13 patients, 6 patients had isolated unilateral caudate infarct, 6 patients had isolated lentiform infarct and one had caudate and lentiform infarct. 6 patients had infarcts due to small vessel disease as evidenced by smaller diameter of infarct and normal cardiac evaluation and carotid Doppler study. 5 patients had infarcts due to large vessel disease as evidenced by abnormal carotid Doppler study. 1 patient had cardiac source of embolism due to mitral stenosis. For 1 patient with migraine, no source of embolism was available.

Though small vessel disease is the common risk factor for asymptomatic basal ganglionic infarcts, we observed large vessel disease in up to 50% of patients as the cause of silent infarct. Toshiyuki et al[62] analyzed the risk factors in silent cerebral infarcts in 219 patients. In his study MRA showed cerebral arterial stenosis ipsilateral to the side of the infarct in 21% of patients.

Carotid artery stenosis[63] was a significant and independent predictor of SCIs in the BG. This finding was consistent with the findings of previous reports. In studies of symptomatic lacunar infarction[64], it has been pointed out that ipsilateral carotid stenotic lesions are potential embolic sources associated with lacunar infarction in the territory of deep perforating arteries. Ghika et al[65] reported that 28 of 100 patients with symptomatic lacunar infarction in the territory of the deep perforators of the carotid system had ipsilateral carotid artery stenosis. Stenotic lesions of the ICA may also play a role in the pathogenesis of lacunes through hemodynamic effects. In an animal model study, diffuse cerebral ischemia from carotid occlusion caused infarction only in the striatum, and a possible toxic effect of dopamine release in the ischemic zone has been assumed to be related to the damage.

Henrik et al[66] analyzed the silent infarction in stroke. He analyzed 500 patients with first ever stroke. 113 (22.6%) patients had 1 silent infarct, 28 (5.6%) patients had 2 silent infarcts, and 6 (1.2%) patients had 3 silent infarcts. None had more than 3 silent infarcts. In total, 186 silent infarcts were detected

in the 147 patients. This community-based study shows that silent infarction in stroke patients is more related to certain stroke risk factors than others and that silent infarction does not seem to influence the prognosis of stroke.

LIMITATIONS OF THE STUDY:

1. Proper follow up for at least 6 months was available only for 23 patients. The remaining patients could not be followed up subsequently. So, We could not ascertain that how many percentage of patients with motor, behavior or cognitive impairment had subsequent improvement and how many were static. So, prognosis was not accurately analyzed in this study.

2. MRI brain with MRA was done only for 18 patients. Remaining patients underwent only CT imaging of the brain. Patients with only CT brain could not be analyzed accurately for the source of infarct. Though extracranial stenosis was diagnosed with Doppler study in patients with large vessel disease, the possibility of intracranial stenosis could not be ruled out in patients with assumed small vessel disease.

3. Though we tried to collect all the cases with basal ganglionic infarcts, there was a tendency to refer all the movement disorder with infarcts and vascular dementia to our study team. So, the true incidence of silent basal ganglionic infarcts may be more than what we analyzed.

CONCLUSION

CONCLUSION

Motor, cognitive and behavioral consequences of basal ganglia infarcts are more common than expected. The current concepts of basal ganglia organization and physiology do not fully explain the disorders observed in man when the striatum and globus pallidus are damaged by crude pathology like infarction.

In our study, dystonia was the most common movement disorder observed in patients with basal ganglionic infarcts, followed by chorea. Mild to moderate depression and apathy is very common in basal ganglionic infarcts; it should be identified and treated effectively to improve the quality of life. Reduced attention span, impaired verbal memory recall and executive dysfunction was the common cognitive impairments identifies which signifies the important role of frontal basal ganglionic subcortical circuitry in higher cognition like executive function and basic cognition like attention and memory.

Patients with caudate infarcts developed more of behavioral and cognitive abnormalities like apathy and executive dysfunction than movement disorders except chorea. Patients with infarcts in the lentiform nucleus developed movement disorders frequently; behavioral disorders were rare in these patients.

While infarcts in the basal ganglia are producing behavioral and movement disorders, many more cases with basal ganglia infarcts are encountered in which similar lesions have no such effects. The reasons for such discrepancies are unknown, but challenge simplistic concepts of basal ganglia motor physiology.

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APPENDICES

LIST OF ABBREVIATIONS

CT	-	Computerized Tomogram
MRI	-	Magnetic Resonance Imaging
OPD	-	Out Patient Department
SLE	-	Systemic Lupus Erythematosus
NPI	-	Neuro Psychiatric Inventory
MMSE	-	Mini Mental Scale Examination
VDRL	-	Venereal Disease Research Laboratory
HIV	-	Human Immuno deficiency Virus
FLAIR	-	Fluid Attenuated Inversion Recovery
TOF MRA	-	Time of Flight Magnetic Resonance Angiography
CSF	-	Cerebrospinal Fluid
PSE	-	Present State Examination
ICA	-	Internal Carotid Artery
PET	-	Positron Emission Tomography
SCI	-	Subcortical Infarct

PATIENT CONSENT FORM

STUDY TITLE: A STUDY ON EVALUATION OF MOTOR, COGNITIVE AND BEHAVIORAL MANIFESTATIONS OF BASAL GANGLIA INFARCTS

Study Centre : Madras Institute of Neurology,
Madras Medical College, Chennai – 600 003

Patient's Name :

Patient's Age :

Identification Number :

Patient may check (✓) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethics committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and to faithfully co-operate with the study team, and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study on Acute symptomatic seizures in the elderly ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological and urine examination. ☐

Signature / Thumb Impression _____ Place _____ Date _____

Patient's Name and Address: _____

Signature of the Investigator: _____ Place _____ Date _____

Study Investigator's Name: _____

PROFORMA

Name: **Age:** **Sex:**

Address:

MIN no:

Complaints:

History of present illness:

Past history: stroke/ TIA/ head injury/ epilepsy/ encephalitis/ psychiatric disturbance/ heart disease/ liver or kidney disease

Personal history: smoking/ alcoholism/ drug abuse

EXAMINATION:

G.E: BP- PR Pallor icterus lymphadenopathy

CVS- **RS-** **Abdomen-**

NERVOUS SYSTEM:

Higher mental function examination:

Attention:

Language: Fluency: comprehension: Repetition:

Naming: Reading: Writing

Memory: Immediate: Recent Remote

Higher cognitive function: Fund of knowledge: Judgement:

Abstract thinking: Similarities: Calculation:

Lobar function test: Frontal lobe:

Parietal lobe:

Temporal lobe:

Occipital lobe:

MMSE:

Addenbrooke's cognitive examination scoring:

NPI score:

Cranial nerves:

Spinomotor system: Bulk: Tone: Power:

Reflexes: Superficial:

DTR:

Sensory system:

Cerebellum:

Extrapyramidal system: Facial appearance: Glabellar tap:

Bradykinesia: Rigidity: Tremor: Postural instability:

Gait: Involuntary movements: Dystonia chorea athetosis

ballismus Dyskinesia

INVESTIGATIONS:

Urine analysis:

Blood sugar - Urea- Creatinine- serum Na - K-

LFT-

Blood T3 - T4- TSH-

Xray chest - ECG- Echocardiogram:

USG abd - HIV serology- Blood VDRL-

CT Brain-

MRI Brain and MRA(if done):

Carotid and vertebral Doppler study:

MASTER CHART

S.No	NAME	AGE	SEX	DEPRESSION	ANXIETY	DISINHIBITION	APATHY	DELUSION	HALLUCINATION	SLEEP DISTURBANCE	COGNITIVE IMPAIRMENT	CHOREA	BALLISMUS	DYSTONIA	PARKINSONISM	CAUDATE INFARCT	PUTAMEN INFARCT	PALLIDAL INFARCT	SUBTHALAMIC INFARCT	NPI SCORE	MMSE	ADDENBROOK'S SCORE
1	MANOHAR	60	M	Y	N	N	Y	N	Y	Y	Y	N	N	N	N	Y B/L	N	Y L	N	34	17	65
2	RAMASAMY	65	M	Y	N	N	N	N	N	N	N	Y R	N	N	N	Y L	N	N	N	10	30	100
3	SONAI MUTHU	58	M	Y	N	N	Y	N	N	N	Y	N	N	N	Y	Y B/L	N	Y B/L	N	15	17	72
4	SARITHAMMAL	52	F	N	Y	N	Y	N	N	Y	Y	N	N	Y H	N	N	Y L	Y L	N	22	27	97
5	ANNAKODI	60	F	N	N	N	N	N	N	N	N	N	N	N	N	Y R	N	N	N	0	30	100
6	KATHIRVEL	48	M	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y L	N	0	30	100
7	MUTHURAJ	62	M	Y	N	N	Y	N	N	Y	Y	Y R	N	N	N	Y L	N	N	N	20	23	83
8	AMUTHA	50	F	Y	N	N	Y	N	N	Y	Y	N	N	N	N	Y B/L	N	N	N	20	27	90
9	SARAVANAJOTHI	54	M	N	N	N	N	N	N	N	N	N	N	N	N	Y R	N	Y R	N	0	30	100
10	THANGAMANI	59	F	Y	Y	Y	Y	Y	N	Y	Y	N	N	Y H	N	Y B/L	Y L	Y B/L	N	38	16	62
11	SIVAKOZHUNDHU	70	M	Y	N	Y	Y	N	Y	Y	Y	N	N	N	N	Y B/L	N	N	N	36	18	63
12	RAMALINGAM	52	M	Y	N	N	N	N	N	Y	N	N	Y L	N	N	N	N	N	Y R	16	30	100
13	DHANALAKSHMI	49	F	N	N	N	N	N	N	N	N	Y L	N	N	N	Y R	Y R	Y R	N	0	30	100
14	NAJIMUNISHA	56	F	Y	N	Y	Y	N	Y	Y	Y	N	N	Y H	Y	Y L	Y R	Y B/L	N	35	19	69
15	ELUMALAI	41	M	Y	N	N	Y	N	N	N	Y	N	N	N	N	Y L	Y L	Y L	N	18	25	68
16	DURAIKANNAN	56	M	N	N	N	Y	N	N	N	N	Y L	N	N	N	Y R	N	N	N	9	30	100
17	SUNDARRAJ	60	M	Y	Y	Y	Y	Y	N	Y	Y	N	N	N	N	Y B/L	N	N	N	44	18	64

S.No	NAME	AGE	SEX	DEPRESSION	ANXIETY	DISINHIBITION	APATHY	DELUSION	HALLUCINATION	SLEEP DISTURBANCE	COGNITIVE IMPAIRMENT	CHOREA	BALLISMUS	DYSTONIA	PARKINSONISM	CAUDATE INFARCT	PUTAMEN INFARCT	PALLIDAL INFARCT	SUBTHALAMIC INFARCT	NPI SCORE	MMSE	ADDENBROOK'S SCORE
18	SANGAIYA	52	M	Y	N	N	Y	N	N	N	Y	N	N	Y HE	N	N	Y L	Y L	N	16	24	85
19	DHARMAR	40	M	N	Y	N	N	N	N	Y	Y	N	N	N	N	Y L	N	N	N	12	25	87
20	PONNUCHAMY	48	M	N	N	N	N	N	N	N	N	N	N	Y HE	N	Y R	Y R	Y R	N	0	30	100
21	UDAIYAL	62	F	Y	N	N	Y	N	N	N	Y	N	N	N	N	Y B/L	N	N	N	18	24	83
22	CHANDRASEKAR	59	M	Y	Y	Y	Y	Y	N	Y	Y	N	N	Y B	Y	Y B/L	Y R	Y B/L	N	42	17	62
23	LATHA	14	F	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y L	N	0	30	100
24	KANNAN	57	M	N	N	N	N	N	N	Y	N	Y L	N	N	N	Y R	N	N	N	12	30	100
25	SANKARALINGAM	55	M	N	N	N	N	N	N	N	N	N	N	N	N	Y L	N	N	N	0	30	100
26	GANESAN	53	M	Y	Y	N	N	N	N	Y	N	N	N	Y H	N	N	Y L	Y L	N	21	30	100
27	VELAYUTHAM	60	M	Y	N	Y	Y	Y	Y	Y	Y	N	N	N	N	Y B/L	N	N	N	48	16	61
28	MANIMARAN	42	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	Y L	Y L	Y L	N	14	25	67
29	MARIYASUSAI	51	M	N	N	N	N	N	N	N	N	N	Y R	N	N	N	N	N	Y L	0	30	100
30	ACHIAMMAL	60	F	Y	N	N	Y	N	N	N	Y	N	N	N	N	Y L	Y L	Y L	N	12	25	72
31	MALLIGA	47	F	N	N	N	N	N	N	N	N	N	N	N	N	Y R	N	N	N	0	30	100
32	PURUSOTHAMAN	58	M	N	N	N	N	N	N	N	N	N	N	Y H	Y	N	Y B/L	Y L	N	0	30	100
33	KARUPPANAN	66	M	Y	N	N	Y	N	N	Y	Y	Y L	N	N	N	Y R	N	N	N	21	23	87
34	MARIMUTHU	50	M	Y	N	N	Y	N	N	N	N	N	N	N	N	Y R	N	N	N	22	27	97

S.No	NAME	AGE	SEX	DEPRESSION	ANXIETY	DISINHIBITION	APATHY	DELUSION	HALLUCINATION	SLEEP DISTURBANCE	COGNITIVE IMPAIRMENT	CHOREA	BALLISMUS	DYSTONIA	PARKINSONISM	CAUDATE INFARCT	PUTAMEN INFARCT	PALLIDAL INFARCT	SUBTHALAMIC INFARCT	NPI SCORE	MMSE	ADDENBROOK'S SCORE
35	THANIGACHALAM	45	M	N	N	N	N	N	N	N	N	N	N	N	N	Y L	N	N	N	0	30	100
36	VELMURUGAN	50	M	N	N	N	N	N	N	N	N	N	N	N	N	N	Y L	Y R	N	0	30	100
37	KAVERIAMMAL	58	F	N	N	N	N	N	N	N	N	Y R	N	N	N	Y L	N	N	N	0	30	100
38	ALAGESAN	55	M	Y	N	N	N	N	N	Y	Y	N	N	Y H	N	Y R	Y L	Y L	N	12	25	84
39	THANDAVARAYAN	60	M	Y	N	N	Y	N	N	N	Y	N	N	N	N	Y L	N	N	N	12	23	81
40	ARULMARY	50	F	N	N	N	Y	N	N	N	Y	N	N	Y H	N	N	Y	Y L	N	8	24	82
41	ALAMELU	71	F	Y	N	N	Y	N	N	Y	Y	N	N	N	N	Y L	N	N	N	18	23	85
42	BALAKUMARI	52	F	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y R	N	0	30	100
43	MURUGESH	58	M	N	N	N	N	N	N	N	N	N	N	N	N	Y R	N	N	N	0	30	100
44	KANNAGI	60	F	Y	N	N	Y	N	N	Y	Y	N	N	N	N	Y L	N	N	N	18	24	85
45	GURUNATHAN	70	M	Y	Y	N	Y	N	N	Y	Y	N	N	N	N	Y B/L	N	N	N	23	23	79
46	CHINNAPONNU	49	F	N	N	N	N	N	N	N	N	N	N	N	N	Y R	N	N	N	0	30	100
47	KATHAMUTHU	46	M	N	N	N	N	N	N	Y	N	N	N	Y U	N	N	Y R	Y R	N	8	30	100
48	SANTHANAM	61	M	Y	N	Y	Y	N	N	Y	Y	N	N	N	N	Y B/L	N	N	N	30	17	63
49	MANI	37	M	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y L	N	0	30	100
50	ANAND KUMAR	42	M	N	N	N	N	N	N	N	N	N	N	N	N	N	Y R	Y R	N	0	30	100
51	KULANTHAIVELU	52	M	Y	N	N	Y	N	N	Y	Y	N	N	N	Y	Y R	N	Y B/L	N	20	23	81

KEY TO MASTER CHART

SEX

M – Male; F – Female

DEPRESSION, ANXIETY

Y – Present; N – Absent

DISINHIBITION, APATHY, DELUSION, HALLUCINATION

Y – Present; N – Absent

SLEEP DISTURBANCE, COGNITIVE IMPAIRMENT

Y – Present; N – Absent

CHOREA

R – Right; L – Left

BALLISM

R – Right; L – Left

DYSTONIA

Y – Present; N - Absent

H- Hand dystonia; HE – Hemidystonia; B – Blepharospasm

CAUDATE, PUTAMEN, PALLIDUM INFARCTS

Y – Present; N - Absent

R – Right; L – Left; B/L – Bilateral

SUBTHALAMIC NUCLEUS

Y – Present; N - Absent

R – Right; L – Left

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. G. Gnanashanmugam
PG in DM Neurology
Madras Medical College , Ch-3

Dear Dr. G. Gnanashanmugam

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled " A study on evaluation of motor, cognitive and behavioral manifestations of basal ganglia infarcts" No. 31012011.

The following members of Ethics Committee were present in the meeting held on 28.01.2011 conducted at Madras Medical College, Chennai -3.

1. Prof. S.K. Rajan, MD	– Chairperson
2. Prof. A. Sundaram, MD Dean i/c , Madras Medical College, Chennai -3	– Member Secretary
3. Prof R. Sathianathan Director , Institute of Psychiatry, MMC,Ch-3	– Member
4. Prof R. Nandhini, MD Director, Institute of Pharmacology, MMC, Ch-3	– Member
5. Prof. Geetha Subramanian, MD,DM Prof. & Head , Dept. of Cardiology, MMC, Ch-3	– Member
6. Prof. Md. Ali, MD, DM Professor & Head ,,Dept. of MGE, MMC, Ch-3	– Member
7. Thiru. T.S. Bharathidasan Administrative Officer, MMC, Chennai -3	– Layperson
8. Thiru. S. Govindasamy . BA.BL	– Lawyer
9. Tmt. Arnold Soulina	– Social Scientist

We approve the Proposal to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee